BRAIN.

PART, IV. 1893.

Original Articles.

ON A CASE OF DESCENDING DEGENERATION OF THE LEMNISCUS, CONSEQUENT ON A LESION OF THE CEREBRUM.

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In spite of the fact that within the last two decades considerable attention has been devoted to the course and connections of the complicated system, or systems, of fibres which are included together under the term *fillet*, or *lemniscus*, and that much has been settled by the study of its development as well as of its degeneration consequent on pathological and experimental lesions, many important points still remain obscure. The records of pathological degenerations are not only very few in number, but, with one or two exceptions, so wanting in details as to the minuter relations, that I am induced to describe the appearances in the medulla, found in a case which I have recently examined, and which seem to throw light on some of the inferior connections of the lemniscus.

The brain from which the specimen was obtained was sent me by my friend, Dr. Ruxton, of Wadsley Asylum, with the note that it had been obtained from an aged female lunatic, who had been for several years completely hemiplegic. The cause of the hemiplegia was an old hæmorrhage which had almost completely destroyed the basal ganglia and the internal capsule on the right

side, and had caused considerable atrophy of the centrum ovale and of the anterior two-thirds of the corpus callosum. The extent of the lesion may be seen in the drawing, fig. 30, plate xi., illustrating my paper on the absence of the corpus callosum in the human brain (*Brain*, 1889, p. 171).

The cerebrum was at that time examined principally with reference to certain points bearing on the results of the congenital absence of the corpus callosum; while the rest of the brain, including the crura cerebri, pons varolii, and medulla, was divided into transverse slices of less than one-quarter of an inch in thickness, and put aside for future investigation. Of these parts, only the medulla oblongata is now available; the other pieces having unfortunately been mislaid. With regard to them, however, it is certain that they contained no gross lesion such as could have given rise to the degeneration to be described. The medulla, which had been carefully hardened in Müller's fluid before reaching me, was embedded in celloidin and cut into thin transverse sections. These were stained by hæmatoxylin according to the Weigert-Pal method, and also by one-quarter per cent. watery solution of aniline blue-black, as recommended by Bevan Lewis.

In the sections which were stained by hæmatoxylin, the right anterior pyramid appeared completely degenerated. Its colour was pale yellow (with the exception of certain blue fibres, to be afterwards mentioned, which traversed it), and its size, as can be seen from the drawings, was greatly reduced. Staining by aniline blue-black showed that there was a slight increase of the connective tissue. In sections made slightly above the plane of the decussation of the anterior pyramid it was easily seen that this was not the only lesion. To elucidate the other degenerations four sections have been reproduced from drawings made with great care by means of Edinger's drawing apparatus in the first instance, into which the details were subsequently filled from the microscope.

Fig. 1 is drawn from a section at the lower extremity of the medulla, immediately above the plane of the decussation of the anterior pyramid, and therefore through almost the lowest fibres which enter into the fillet.

On the right side the compact bundle of internal arcuate fibres which arises from the nuclei of both divisions of the posterior columns of the cord is seen to pass forwards, in the normal manner, as a compact, somewhat arched bundle of fibres. This, after passing outside the ascending root of the glosso-pharyngeal nerve (seen as a small mass of transversely divided fibres), turns inward and crosses the mesial plane as the decussation of the fillet (D.F.) to insert itself between the antero-lateral ground bundles (A.L.G.B.), and the anterior pyramid. (The distance to which these fillet fibres pass outwards between the two last-mentioned systems should be noted.)

On the left side these arcuate fibres are very few in number, and, at first, do not attract the eye. Those that are here present seem to be of finer calibre than those on the other side. This may, perhaps, be owing to the fact that they are few in number and separate from each other. They almost all enter the nucleus cuneatus (N.C.); of hardly any can it be said definitely that they join the nucleus gracilis (N.G.). After crossing the mesial plane at D.F. they appear between antero-lateral basis bundles and the degenerated anterior pyramid (Pyr.), with the exception of a few fibres which pass as anterior external arcuate fibres (F.A.E.A.) round the inner and ventral aspect of the pyramid (the course of which will be referred to below). Both the nucleus gracilis and the nucleus cuneatus on the left side are very considerably less than the corresponding nuclei on the right side. At this level I think that the nucleus gracilis has atrophied to a rather greater degree than has the cuneate nucleus.

The two divisions of the posterior columns seem to present a normal appearance on both sides, as also do all the other structures, with the exception, already mentioned, of the pyramids (Pyr.). There was no detectable difference in the size of the ascending roots of the 5th nerve, the substantiæ gelatinosæ (S.G.), the direct cerebellar tracts (D.C.T.), the antero-lateral ground bundles (A.L.G.B.), or the anterior external arcuate fibres (F.A.E.A.). The asymmetry of the two sides of the section is due to the shrinkage of the degenerated right anterior pyramid, and of the lower end of the fillet, which has disappeared from the spot immediately behind it.

Fig. 2 is drawn from a section made through the lower extremity of the inferior olive. On the right side, the nucleus gracilis and nucleus cuneatus are of normal size. The median internal arcuate fibres (F.A.I.M.), which emerge from these, pass forwards and inwards in curves with their convexity outwards. They may be almost divided into two groups, a posterior and an anterior. The posterior group forms a bundle of fibres nearly as compact as in fig. 1. Some of these pass close round the inner side of the ascending root of the glosso-pharyngeal nerve (and indeed seem to arise from it). This group of fibres passes through the more posterior fibres of the posterior longitudinal fasciculus, crosses the raphe at a somewhat acute angle, and passes forwards and slightly outwards. The second or anterior group of these internal arcuate fibres passes forward through the formatio reticularis in more open curves of somewhat widely separated fibres, which cross the raphe and enter the fillet (or inter-olivary stratum) of the opposite side.

On the left side the two nuclei of the posterior columns are atrophied, apparently to nearly equal degree (though on the whole, I think that the nucleus gracilis is the more affected). The fine felt work of fibres within these nuclei has to a large extent disappeared, so that their colour in hæmatoxylin sections is decidedly paler than those on the right side. The median internal arcuate fibres have almost entirely disappeared from this side. Those which pass to the inner side of the ascending root of the glosso-pharyngeal nerve have all but entirely gone. Of the others, a small number of separate (and fine) fibres arch along the dorsal part of the formatio reticularis, and cross the posterior longitudinal fasciculus to enter the inter-olivary stratum of the opposite side. The comparison of the formatio reticularis in the intermediate field of Flechsig on the two sides is most striking. On the right the arcuate fibres form a distinct tract, which catches the eye at once, on the left there remain but a few fibres which require a careful search to be seen. The contrast between the two sides of Flechsig's inner field, viz., that between the raphe and the root of the hypoglossal nerve (not figured) is no less striking, though perhaps not quite so much so as in figs. 3 and 4. The strip of transversely divided fibres which extends

along the right side of the raphe from the hypoglossal nucleus to the degenerated anterior pyramid is greatly reduced in size. This change is most marked anteriorly, where the fibres become attenuated to a narrow point (Fill.) which projects into a pale V-shaped area, the outer limit of the V being the internal accessory olive; and the inner, the area from which the degenerated fibres of the fillet have disappeared. The most dorsally situated fibres in the area form a band considerably less broad than the corresponding ones on the other side. The anterior external arcuate fibres which are seen to arise from the fillet, pass, partly as a small strand between the inferior olive and the degenerated pyramid, partly round the inner and anterior aspects of the pyramid, at the outer side of which it joins the first strand. A third series passes as scattered fibres into the pyramid. I have been unable to detect any diminution in the number of any of these three strands of external arcuate fibres on careful comparison of the two sides. Beyond the asymmetry of the medulla, which results from the above-mentioned degenerations, no other abnormality could be seen in the section.

Fig. 3 represents a plane of section very slightly higher than that of fig. 2, but still through the lower end of the olive. The asymmetry of the two sides of the section is as marked as in fig. 2, and its cause is the same. The all but complete disappearance of the internal arcuate fibres on the left side is as marked as in fig. 2. Those fibres which remain are fine and separate from each other. They lie almost entirely in the posterior part of the formatio reticularis, and terminate mainly in the internal cuneate nucleus, but also partly in the nucleus gracilis. The cuneate nucleus appears in this section to show a relatively rather higher degree of atrophy than does the nucleus gracilis. Both are much paler than normal, owing to the disappearance from their interior of the terminal twigs of the arcuate fibres. In Flechsig's inner field the contrast between the two sides is greater than in fig. 2. The fibres in this region which lie between the hypoglossal nucleus and the anterior pyramid are usually divided into two pretty nearly equal parts, a line drawn between the two dorsal ends of the internal accessory olive forming the supposed boundary line. The portion of fibres behind this

imaginary line is usually regarded as the posterior longitudinal fasciculus, that anterior to it as the fillet (or inter-olivary stratum). Both these strands are reduced in breadth, but to different degrees. The posterior half is about one-half the width, the anterior is about one-eighth or less, being fined away to a narrow point anteriorly, so that the internal accessory olive comes almost to touch the raphe. A fine pale line is seen along the outer side of both halves of the strand, showing where the degenerated fibres have fallen out. The hypoglossal nerve on the right side has approached the raphe considerably, being only about half as far from it as the left nerve.

The external anterior arcuate fibres which lie in front of and behind the right anterior pyramid, are in no way fewer than those of the opposite side. The section was otherwise perfectly normal.

Fig. 4 was drawn from a section made slightly above the middle of the olive, and just above the upper end of the nucleus gracilis. On the left side those median internal arcuate fibres which pass from the cuneate nucleus behind the inferior olive, have almost entirely disappeared. A few can still be seen to enter the internal cuneate nucleus (N.C.I.), but those which, on the right side, pass in relation to, and as if arising from, the ascending root of the glosso-pharyngeal nerve (IX. Asc.) have entirely disappeared. On the right side all the median internal arcuate fibres are normal. The lateral internal arcuate fibres which pass from the left restiform body, partly through, partly behind, and partly in front of the olive, to end in the opposite olive, are equally present on the left as on the right side. The great contraction of the fibres in the whole extent of the inner field of Flechsig is very distinct, being marked off from the rest of the formatio reticularis by a narrow pale band, which widens out anteriorly, so that in the drawing it is difficult to distinguish it from the internal accessory olive. The posterior part, which corresponds to the posterior longitudinal fasciculus (P.L.F.), is, on the right side, only one-half the breadth of that on the left; while the inter-olivary stratum (Fill), is reduced to one-eighth of the breadth of the other. Those fillet fibres which are found between the internal accessory and the main olive, have also disappeared on the right side. The anterior external arcuate fibres (F.A.E.A.)

are quite normal on both sides. The two restiform bodies (C.R.) are quite equal, and the posterior external arcuate fibres (F.A.E.P.) are in no way diminished on the left side. There is no other abnormality in the section.

This case shows that, in consequence of a lesion destroying the basal ganglia and the internal capsule in the cerebrum, the fillet may degenerate in a descending direction, through the inner field of Flechsig in the same side of the medulla, through the median internal arcuate fibres of the opposite side, as far as the nuclei of the two divisions of the posterior columns of the cord.

The degeneration in the inner field is not co-extensive with what is usually regarded as the inter-olivary stratum. This name is given to those longitudinal fibres which are contained between the raphe and the inferior olive, and between the posterior surface of the medulla and an imaginary line joining the hinder limits of the two internal accessory olives.

The degeneration affects about seven-eighths of the fibres in this area, those fibres which remain intact forming a very narrow zone along the side of the raphe. But the degeneration also extends in a backward direction as a very narrow zone along the outer side of the posterior longitudinal fasciculus. From the examination of several other cases of degeneration in the medulla, I can find no evidence to show that these fibres do not belong to the fillet, and I am inclined to think, therefore, that the limits of the fillet are more extensive than developmental evidence would appear to show.

Those fibres which remain in the anterior part of the inner field are either part of the system of the posterior longitudinal fasciculus, or are fibres of the fillet which have escaped degeneration, either because they degenerate only in an upward direction, or, if they degenerate downwards, because they arise in the pons and corpora quadrigemina, from levels too far down to be affected by a lesion in the cerebrum. We shall see reason to believe that the latter is the case. It is possible that this may be the explanation, but it would be of great importance to compare this case with one where a descending degeneration had followed a pontal lesion, and where presumably both the cerebral and pontine fillets had been cut across. In Spitzka's case (*loc. cit.*) the whole

stratum would appear to have degenerated, but his figures are somewhat too small to enable one to form a definite opinion from them.

Certain of the internal arcuate fibres of the opposite side have disappeared. The lateral group which connects one olive with the opposite restiform body, are quite intact; but the great majority of the median arcuate fibres which are found in the lower part of the medulla have disappeared. A comparatively small number remains. These are seen as fine fibres, extending from the raphe along the most posterior part of the formatio reticularis, towards both nuclei of the posterior columns.

At the lower part of the medulla the number increases, till a compact bundle is formed, which can be traced pretty definitely for the most part from the undegenerated remnant of the opposite fillet into the nucleus cuneatus. It would appear from this, therefore, that the nucleus cuneatus is more directly connected, than is the nucleus gracilis, with those fibres of the fillet which end below the cerebrum. This is in harmony with the results obtained from the embryo (see Illustrations of Mid and Hind Brain, plate xiii., fig. 3, where it is seen that the earliest fibres of the fillet arise from the cuneate nucleus, and cannot be traced higher than the pons; see also reference to Bechterew's paper on the Fillet at page 37, which agrees with this view). It is certain, however, that all the persistent arcuate fibres do not arise from the fillet, but that a large number of them are directly continuous with the opposite anterior external arcuate fibres, which pass in undiminished numbers to the opposite restiform body, partly in front of, partly through, and partly behind the anterior pyramid. Hösel says that in his case they have entirely disappeared from within the pyramid. I have carefully compared my sections with others from a simple degeneration of the pyramid, and can find no difference in the numbers of undegenerated fibres within the pyramids in the two cases. It is possible that Hösel has over-estimated the number of these fibres in the sound side, as it is likely that the wavy appearance, noted by him as due to the passage of arcuate fibres through the pyramid, is largely the result of a re-arrangement of the pyramidal fibres, preparatory to their decussation. It should be noted also that

Fig I

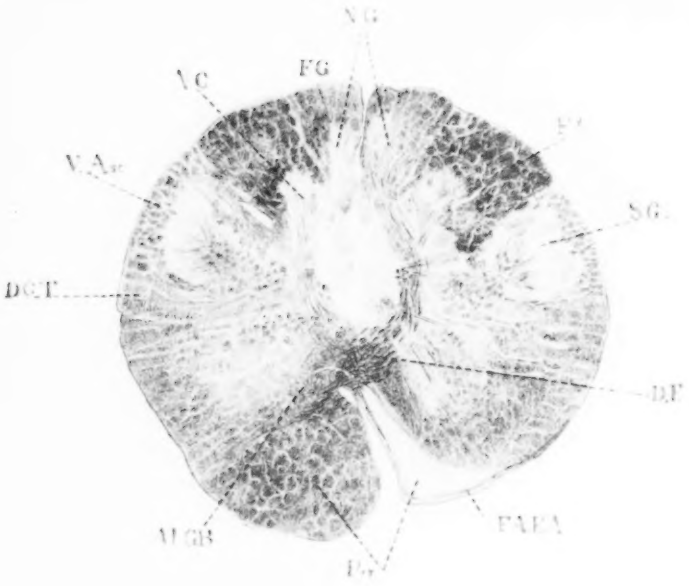

NG
VC
FG
V.As
SG
DCT
BF
VGB
FAEA

Fig II

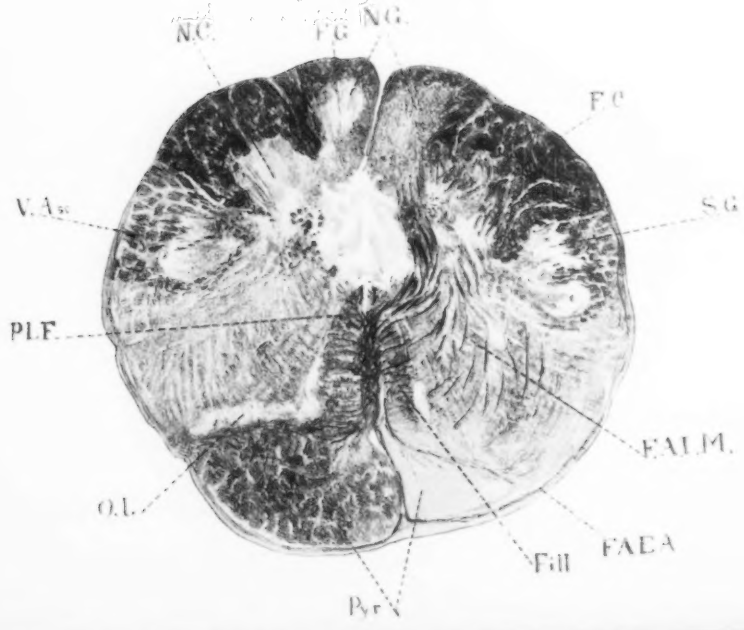

NC
FG
NG
FC
V.As
SG
PLF
FALM
O.L
FaB
FAEA
PyF

Fig. III

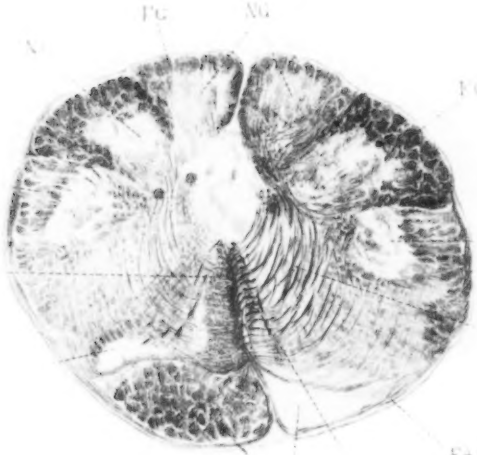
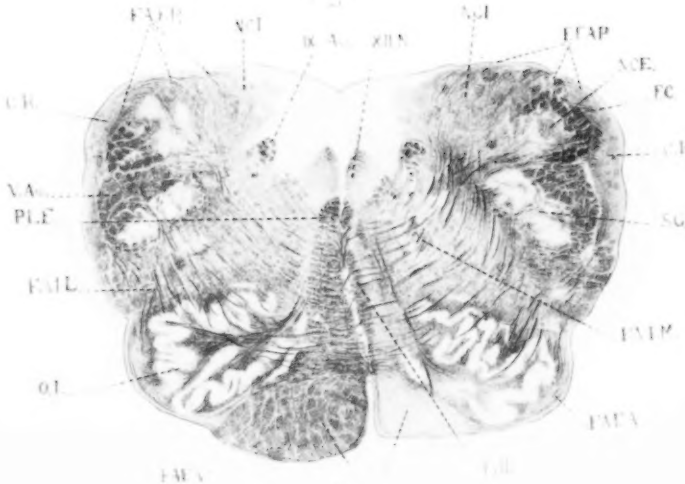


Fig. IV



those internal arcuate fibres which pass from the inner side of the ascending root of the 9th nerve, have all but disappeared, while the nerve itself is quite normal. Through the kindness of Dr. Abernethy I have had the opportunity of comparing with this case one of degeneration of the ascending root of the 9th nerve, in which these arcuate fibres are intact. It is, therefore, certain that the ascending root of the glosso-pharyngeal nerve is not directly connected with the fillet.

Both nuclei of the posterior columns showed a considerable degree of atrophy on the left side. This was evidenced, in the hæmatoxylin section, by a decrease in the size of the nuclei, and a diminution of the network of fibres belonging to the internal arcuate system; and, in sections stained with aniline blue-black, by disappearance of the nerve cells and also by an increase of the connective tissue. At the lower extremity of the medulla, the nucleus gracilis seems to have atrophied to a greater degree than the nucleus cuneatus; but at levels opposite the upper extremity of the nucleus gracilis the internal cuneate nucleus seemed to have degenerated to an equal, if not to a greater, extent. These facts would appear to warrant the following conclusions as to the connections of the fillet: that the nucleus gracilis is the terminal point for the great majority of the fibres which pass upwards to the cerebrum, while the nucleus cuneatus, at its lower part, receives mainly fillet fibres from the pons, which have escaped degeneration, but, in its upper part, getting also fibres from the cortical fillet. Observers of the degeneration of the nuclei have obtained somewhat divergent results; Monakow finding in an experimental case that the nucleus gracilis was the more atrophied, while Déjérine, Flechsig, and Hösel found the greater change in the cuneate nucleus. Spitzka, in his case, where the lesion was in the pons, and where, presumably, all the fibres of the fillet were degenerated, found the nucleus gracilis the more affected. The external cuneate nuclei are unaffected, and are therefore, presumably, not end stations of the fillet at all.

It is remarkable that none of the other tracts connected with these two nuclei show any evidence of diminution in size. Both divisions of the posterior columns of the two sides are equal, so, too, are both sets of posterior and anterior external arcuate fibres.

It follows, therefore, either that there are in these nuclei still undegenerated cells, which are the trophic centres for these arcuate fibres, or else, that their trophic centre is situated in the cerebellum. My case does not enable one to draw any conclusion as to this, which can only be proved by a comparison of destructive lesions in the nuclei or in the middle lobe of the cerebellum, the two terminal points of these strands. It is known that the posterior columns do not degenerate downwards.

Unlike Hösel's case, no difference could be traced between the two 5th nerves.

The most important cases of descending degeneration from a cerebral lesion are those of Witkowski (*Arch. f. Psychiatrie*, Bd. xiv., p. 140); Déjérine (*Arch. de Physiol.*, 1890); Hösel (*Arch. f. Psychatrie*, Bd. xxiv., p. 452); Monakow (*Neurol. Centralblatt*, 1885, p. 69). Spitzka, in the *Medical Record*, 1884, p. 393, describes carefully a descending degeneration from a lesion in the pons. Most of the other cases, which are fully referred to in Déjérine's and Hösel's papers, are too briefly described to be of much value.

The four figures are photogravures (by Messrs. Thevoz, of Geneva) of the original pencil drawings. Fig. IV. is reduced in scale ($\frac{8}{11}$).

REFERENCES IN FIGURES.

A L G B, antero-lateral ground bundles.
C R, restiform bodies.
D C T, direct cerebellar tracts.
D F, decussation of fillet.
F A E A, antero-external arcuate fibres.
F A E P, postero-external arcuate fibres.
F A I M, medio-internal arcuate fibres.
Fill, fillet (not lettered in fig. 3).
N C, nucleus cuneatus.
N C I, internal cuneate nucleus.
N G, nucleus gracilis.
O I, inferior olive.
P L F, postero-longitudinal fasciculus.
Pyr, anterior pyramids.
S G, substantia gelatinosa.
V.Asc., IX.Asc., XII.N., v., ix. and xii. nerves.

A THEORY OF CORTICAL VISUAL REPRESENTATION.

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BEFORE attempting any theory of the representation of vision in the cerebral cortex that might explain all the opposing facts, it would seem necessary to enumerate a few of the most important, the best authenticated, and it would also appear *pari passu* most contrary of the results of and deductions from experiments on animals in this relation. For while, like all other kinds of cerebral localisation, that of the cortical centres for vision owes its discovery to experimental investigation, in this particular branch of this method of research so many different inferences have been drawn from results which in themselves have been of such varied and often contrary nature, that even after a careful study of the literature of the subject it is difficult to decide which, if even any, are now regarded as conclusive and indisputable.

And first as to the mere extent of the visual area. It would seem that in the opinion of many this comprises both the angular gyrus and the occipital lobe, for Ferrier states "in order to cause complete and permanent loss of vision in both eyes it is necessary completely to extirpate both angular gyri and both occipital lobes" ("Functions of Brain," p. 272), and again that "complete destruction of this area (the occipito-angular) in the one hemisphere causes permanent hemiopia to the opposite side . . . . while bilateral destruction causes complete and enduring blindness of both eyes" ("Cerebral Localisation," p. 40), where he goes on to state (pp. 46-48) that Tamburini and

Luciani too include the angular gyrus in the visual area; and Schäfer and Horsley also appear to believe that the angular gyrus is to some extent concerned in vision as well as the occipital lobe.

To many an unbiassed view of Munk's own account, from his description of the phenomena noticed after lesion of this part, would lead to the same inference, though he himself professes to confine the visual area to the occipital lobe alone. Certainly, the movements of eyeballs and pupils, and sometimes of the head, which occur on stimulation of this region, and which, considering the length of the latent period, can only be interpreted as reactions to a sensory stimulus, supports the view of the area's function; so that I think we may fairly say that there are good grounds, without discussing the clinical side of the question, for believing that the cortical visual area probably includes the angular gyrus as well as the occipital lobe.

Considering each of these two regions separately; as to the occipital lobe, it is well known that many observers—Schäfer, Munk, Horsley and Schäfer, Sanger Brown and Schäfer, Erlman, Thompson and Sanger Brown, with others—describe a condition of homonymous hemiopia from paralysis of correlated halves of the same-named sides of the retinæ, after cortical lesion of this alone, and some profess to show blindness following a bilateral lesion. On this point there is not agreement, however, for Horsley and Schäfer also show that after removal of both occipital lobes some vision remains, and though a large amount of clinical evidence has been brought to prove the pre-eminent visual function of this lobe, still the fact remains that many cases, also of lesions of the occipital lobe, have been reported without any observable symptoms (as instanced by Ferrier's "Localisation of Cerebral Disease,") and though, if unilateral, this might be accounted for from the hemiopia being unnoticed or overlooked—as, indeed, it often is in actual clinical practice — still, as these cases include some of bilateral lesions when the patient should theoretically have become blind, it is obvious that some other explanation is necessary. Schäfer himself (BRAIN, vol. x., p. 372) states

that a lesion to produce permanent hemianopia from injury to the occipital lobe probably must include some at least of the cortex of the angular gyrus, and this would agree with Ferrier's hypothesis that by so doing a portion at any rate of the angular radiations are involved, and I hope to be able to show that for the blindness *per se* seen after bilateral lesions this is probably the explanation.

Ferrier has laid it down that "even after most extensive destruction of the occipital lobes no portions of the retinæ appear to be absolutely blind," by which I conceive that the animals still retained some vision, and he also states ("Functions of Brain," p. 273) that the occipital lobes *per se* "can be injured or cut off bodily almost up to the parieto-occipital fissure on one or both sides simultaneously without the slightest appreciable impairment of vision."

Lannegrace (*Archives de Médecine Expérimentale*, 1889) has also published results of experiments in which he too failed to obtain the impairment of vision spoken of by other observers, so that it is evident, without referring to the clinical side of the question, which will be returned to later, that some observers still doubt the occipital lobe being at any rate the sole and only cortical area for vision, but that there is much evidence in favour of it taking a large share seems certain.

As regards the angular gyrus alone, Ferrier has from the first laid it down that unilateral lesions of the angular gyrus cause temporary blindness in the opposite eye, giving at the same time reasons for believing that the eye of the same side was simultaneously, but to a much less extent, affected. Also that bilateral lesions caused blindness of both eyes, though this too was only of a temporary character.

Lannegrace also found crossed temporary amblyopia to follow a lesion of this area, and Schäfer (BRAIN, 1888, p. 159), after destroying both angular gyri found that blindness for a few days followed. So that it would appear that the angular gyrus is also credited with a considerable share of cerebral visual perception, though in this case of a totally different character to the function of the occipital lobe.

As a result of all these conflicting observations several equally conflicting deductions have been generally accepted.

(1) That the visual area on each side comprises both angular gyrus and occipital lobe.

(2) That hemiopia follows lesion of the occipital lobe, (*a*) according to some, only when associated with damage to fibres from the angular gyrus; or, according to many, (*β*) after lesions of this lobe alone and of that only, Schäfer, as is well known, in his scheme also providing for a (*γ*) sectional representation of the visual field, in the occipital cortex.

(3) That in the angular gyrus of each side there is a "higher" representation of the whole of the opposite visual field, (*a*) and to a smaller extent of that of the same side (Gowers). (*β*) That this area (angular gyrus) probably, as Ferrier has shown, corresponds to that for clearest vision, *i.e.*, central vision as subserved by the macula lutea.

It will, therefore, be evident that these facts and theories necessitate that whatever scheme would really explain and reconcile all these differences must account for the following:—

(1) The angular gyrus and the occipital lobe being both concerned.

(2) Amblyopia of a temporary character following experimental unilateral lesions of the angular gyrus, and bilateral lesions of the same producing blindness also of a temporary character.

(3) Unilateral occipital lobe lesions being followed at some hands by homonymous hemiopia and bilateral lesions by blindness; while at other hands, unilateral and bilateral were both equally productive of negative results.

(4) Smaller occipital lesions showing instead of complete hemiopia some sector defects.

(5) The evidence in favour of the angular gyrus subserving central vision, *i.e.*, the area of clearest vision and the yellow spot.

Now there is one theory of cortical visual representation which it seems to the writer would account for all the perplexing contrary experimental results, would reconcile

these several conditions, and would also agree with several clinical facts hitherto difficult to account for.

I would venture to submit that vision in the general area of the retina is distinct from that of the macula lutea, *i.e.*, that though both may frequently, and, indeed, usually may, act together, vision is possible by the retina alone without the macula lutea; or, on the other hand, by the macula lutea alone without the surrounding retina. For it is laid down and generally accepted that for clear sight, putting aside the question of the size of the visual angle, the state of the cornea, lens, vitreous, and amount of illumination and pupil area, the conditions are that the image, as well as being focussed on the retina, must be formed at the centre of the yellow spot, and it will not, I think, be denied that it is the yellow spot that subserves any and every occasion for clearest and most distinct vision. But given a perfectly normal macula lutea in ordinary use, it is almost equally certain that the remaining area of the retina, even allowing that the extreme outer periphery may not do so (Landolt), also subserves vision; as to this fact it seems almost needless to bring evidence, but there is certainly a marked loss in such diseases as syphilitic, hæmorrhagic and pigmented retinitis, the defect of sight being often in direct ratio to the amount of retina affected—until only the central area subserved by the macula lutea may remain, and it is here worthy of note that vision may be exceedingly good in a bright light when only this portion remains to see with.

On the other hand, axial neuritis and tobacco atrophy may both cause a distinct loss of central vision with a well-marked central scotoma, but still a very fair amount of vision is retained by the unaffected surrounding retina. Indeed, in this particular I would remark that a central loss of sight may be quite unperceived for some time. We are none of us, I think, aware of the blind spot normally present in our retinæ, and the central scotoma of tobacco amblyopia is occasionally overlooked by its possessor. A good deal, perhaps, depends on the intelligence and power of observation of the patient, which, however, does not

affect the fact, so that we may allow that vision is possible with loss of the centre of the field if there is retention of the periphery, and that it is equally possible with the periphery lost or damaged and the centre remaining. It is probable that most persons, unless for, if I may so term it, loose general vision, use the macula lutea, chiefly if not exclusively for vision requiring any attention or application, and it would presumably follow that its loss would be very evident. Now, I would therefore suggest that the true explanation of the discrepancies of experimental, and indeed clinical evidence lies in the fact that there is a separate and distinct representation of the macula lutea and the correlated retinal halves in the cortex, namely, that the macula lutea and that only is represented in the angular gyrus, chiefly of the opposite side, but to a smaller extent of the same side also; the representation being the bilateral one enunciated by Ferrier ("Cerebral Localisation," p. 58), and now largely accepted; while each occipital lobe serves for the representation of the correlated halves of the same named side of each retina, but without the macula lutea of either, which is represented solely in the angular gyrus, as before said.

On consideration as to how this theory would account for the opposing results of experiments, &c., taking first the most striking, *i.e.*, the negative effects of occipital lobe lesions, in accordance with this scheme, a lesion strictly confined to the occipital lobe would then cause a hemiopia to the opposite side, but there being still distinct vision in both maculæ luteæ, which certainly in animals is the portion of the retina chiefly used in perception of objects, &c., by which the animal's sense of sight is generally tested in the laboratory, the fact of there being much loss or any could easily escape observation; and even in a bilateral lesion given it still strictly confined to the occipital lobe even then blindness would not result, both maculæ luteæ still remaining, while on the other hand the extreme ease with which the angular radiations might be injured in the experiment would account for the very complete hemianopia so distinctly described by many observers, and

a fortiori for the blindness they have seen to follow bilateral injuries.

The occipital cortex, in my opinion, particularly subserves crude binocular vision; and I think this lobe will be found developed in animals in direct ratio to the amount of binocular vision they possess, while the angular gyrus is the "higher" field of representation of particular clearest and central visual perception. It is not, of course, pretended for a moment that the macula lutea cannot and does not subserve binocular vision; it undoubtedly does, but that is not its essential function, the remainder of the retina, it is maintained, being the essentially binocular mechanism.

In regard to the amblyopia seen after experimental lesions in the angular gyrus, the macula lutea and that part only being represented in this area in such a manner that each angular gyrus subserves the macula lutea of the opposite side, and to a less extent of its own, and this being in animals as in men such an important part of the visual field, the disturbance resulting from a sudden loss of it such as would result from an experimental lesion would, I think, satisfactorily account for the temporary amblyopia that has been seen to follow it, and the fact of its being only temporary would, it is obvious, be accounted for by its bilateral representation, and perhaps to a smaller extent by the gradual use of the peripheral retina, which latter fact would be the explanation of the non-permanence even of bilateral lesions.

From an anatomical point of view a scheme to represent this theory could easily be given, for, in addition to the crossing in the chiasma of the fibres from the inner half of each retina, all that would be required would be to show the fibres from each macula lutea also crossing in the chiasma with these and going with them, and the temporal fibres of each side after communication with the optic thalamus, the internal geniculate and anterior quadrigeminal bodies, as the optic radiations, separating and passing to their several distributions in the angular gyrus and the occipital lobe shortly after. But to account for the double representation in the angular gyrus almost un-

doubtedly present, some fibres from each macula lutea must also be continued in the optic tract of the same side to the cortex, and though another possible arrangement in place of this, *i.e.*, that of fibres being continued along the same side, is that which has been supposed in nearly all the schemes hitherto brought forward, namely, that a re-crossing of some fibres at least takes place in the mid-visual centres (presumably the quadrigeminal or geniculate bodies, for the fibres going to the optic thalamus almost undoubtedly subserve "hemiopic" vision), still, it is generally stated that the decussating fibres over the anterior quadrigeminal bodies crossing the aqueduct, if not solely, are largely derived from the fillet, others from the "central connections of the descending root of the trigeminus" (Obersteiner); and though the posterior quadrigeminal bodies are almost continuous with one another in the middle line, still these latter have been generally believed not to be related in any way to vision *per se*, and as I am certainly not aware of any one having shown a decussation of the geniculate bodies it would seem as though this arrangement in the chiasma were the only possible one, although at the same time the bilateral representation could be more easily understood by a division of the fibres after their junction with cells (*i.e.*, that cells receiving one apparent process could give off two, one to be reckoned again for distribution to the opposite side), than it can be understood how a double set of fibres should go from identically the same area in the macula lutea.

It seems needless to remark that this is not a solitary example of a bilateral representation in the cortex—the same being seen so conspicuously in that of the trunk muscles, respiratory, laryngeal, and all these habitually used in concert, and though it might appear strange that so small a portion of the retina or the macula lutea should have a separate representation in this way, it may be noticed that apart from the functional importance—from the extreme tenuity of the fibres an extremely large number are distributed to this area. Bunge (quoted by Nettleship, p. 199) gives a case of central scotoma, in which though only one-

fortieth of the field was lost, yet one-fourth of the whole of the fibres were degenerated. There is probably, too, a difference histologically as well as in number between the fibres of the retina and the macula, for it has been repeatedly shown that fibres from some part of each optic nerve pass into both tracts, the probability being that these are from the fovea, and attempts have been even made to show that degeneration which has been traced into both tracts is from this area. Gowers (vol. ii., p. 142) seems, while doubting this latter particular, to take it for granted that fibres from each macular region pass to both hemispheres, but does not give any details; and Obersteiner ("Anatomy of Central Nervous Organs," p. 284), while he says "that each visual centre is connected in a partially crossed manner with both eyes," also does not explain how this takes place; certainly no one has ever even suggested that the "hemiopic" fields are ever doubly represented, *i.e.*, that any part from the same named halves are represented in both hemispheres, so that it is altogether more feasible to suppose these double fibres to be derived from the central fovea, and it is just possible that the "direct cortical root," that is to say, the deep root, of fibres branching off from the tract to the crus in front of the geniculate bodies, and which, as Obersteiner remarks, probably contains fibres from both optic nerves, is the path of these macular fibres to the cortex. If this were so, one would expect to hear of direct degeneration between the angular gyrus down this tract to the optic nerve, or *vice versâ*, there being no cells (?) midway to break the process. As to the functional representation of the angular gyrus and occipital lobe respectively, it seems probable that the representation in the angular gyrus is as of a whole, *i.e.*, is not a functional division into segments, so that any partial lesion would only cause a partial lowering of the whole functional capacity and a relative loss; whereas in the occipital lobe it seems most probable that there *is* a segmental functional division; whether according to Schäfer or Munk's scheme, to which or to either, is not clear. Arguments have been brought forward that the cases adduced to prove this clinically are always examples of implication of the optic

radiations and not of disease of the cortex, and though this fact in these instances is more than probable, still, it is difficult to understand why differentiated optic radiations should exist just under the cortex if not for distribution to different parts of that cortex, it being admitted that these different radiations are in relation with segments of the retinal area. A careful consideration of defects of sight from a clinical point of view show facts which agree with this theory as to the separate representation of the centre and the periphery of the retina in the cortex of the hemispheres—facts which may also serve to explain certain points in connection with them hitherto of very debatable character.

It was first shown by Willbrand as a distinguishing mark between tract and central hemiopia, that a pencil of light thrown on the blind side of the retina failed in the former variety to cause contraction of the pupil. But Ferrier ("Functions of the Brain," p. 294) has also shown that all these cases have the fixation point cut by the dividing line. Now everyone is aware of the extraordinary difference in many cases of hemiopia, whether the dividing line passes through the fixation point, or whether this is included in the seeing half (Willbrand puts the variation as 23 in 50), that is, whether the macula lutea region is divided or not. Gowers ("Manual," vol. ii., p. 142) tries to account for this by doubting whether the fixation point is ever actually divided, but it must be obvious that according to his argument when the line is, as he shows it, so extremely close to the fixation point in division, the probability of an error is enormously increased. I mean that if a patient first denies seeing beyond the fixed area, and then, being brought down to the division of a minute point, inclines to think that he sees it—considering the difficulty at all times of keeping the line of sight motionless—it is extremely probable that his first view was the correct one.

I would, therefore, submit that the true explanation lies in the distribution of area suggested, namely, that in the tract, of course the macular fibres still flowing with those of the other part of the retina; a hemiopia due to lesion here

which would necessarily include these too would not show a retention of the centre in the seeing half of the field. Whereas, after the individual centres—the optic thalamus, the geniculate, and quadrigeminal bodies, the macular fibres going to the angular gyrus diverge from the retinal ones proceeding to the occipital lobe, and therefore an affection directly in or under the cortex of the latter would cause a hemiopia, in which the fixation point was included in the half in which vision was still retained. I am aware that it would seem at first that if this explanation holds, then where the fixation point is included in the blind half that there should be a small projection of it into the seeing portion to agree with the fact that the macular fibres are not divided, but decussate as a whole. I have not had an opportunity lately of testing this point, but I do not think the line is ever perfectly straight, and the area is so small that it might easily escape observation. At the same time it must be remembered that if the lesion were sufficiently low down, near the optic tract, where presumably the macular fibres would be only just leaving the company of the others if they were not indeed still in conjunction, the hemiopia would simulate that due to disease of the tract itself. This would account for the apparent variability of their symptoms and for Gowers' dictum that the difference in the fixation point does not depend on the situation of the lesion. I would urge that it most certainly does, and that a careful examination of cortical and subcortical cases of hemiopia where the angular gyrus is not involved, will always show that the fixation point is in them included in the seeing half. Several of the figures Gowers himself gives, and which presumably are of cortical disease, show this, viz., figures 75, 76, 77, 80. I have never seen a segmental defect illustrated (which is, *par excellence*, usually supposed to be due to a lesion of the occipital lobe, and if not cortical, subcortical) in which the central area was represented as blind, which is a fairly strong argument

In those cases where the fixation point is divided, and yet the lesion is not in or near the tract, I think it will be found that there is generally at the same time a peripheral contraction of the remaining half fields, and that, as might

be expected, there is usually some implication of the fibres radiating from the angular gyrus.

The clinical cases of occipital lesions in which there were no visual symptoms would probably be all explained by the assumption before given for corresponding experimental results, that the angular gyrus being unaffected some sight did actually remain. But still, there is very strong clinical proof of the association of occipital lobe disease (either alone or plus that of the angular gyrus) and hemiopia, cases even of amaurosis after bilateral lesions having been reported. As to there being any evidence of actual cortical disease producing such is another matter. I had occasion to examine carefully the original records of all the recorded cases of occipital and parietal lobe lesion up to June, 1890, and failed to find a single case of cortical lesion strictly limited to the occipital cortex causing hemiopia. Nearly all the many described as coming under this category, even when they were not due to or accompanied by a tumour or compressing lesion in the medullary substance, in which case, for localisation purposes, they are of course absolutely valueless, even when not so were each accompanied by so many other lesions in the temporal hippocampal or parietal cortex as to render it absolutely impossible to say that the symptoms were due to affection of the occipital lobe alone. It has also been strongly argued by Seguin and Nothnagel that the cuneus is the ultimate seat of hemiopic visual perception, and Obersteiner in his recent work also places this as the culminating point in the visual scheme. It may be so, in which case it is difficult to see the utility of so large a remaining portion of the occipital lobe, but the cases brought in support of this theory are open to the same objections before alluded to, and I am not aware that any one has ever proved the optic radiations to end at this particular point, although it is most probable that the implications of these radiations is the explanation of these cases, and also of that theory (Exner) that would substitute the apex for the cuneus. In monkeys too it may be remarked the cuneus may be excised without appreciable result as to vision.

As regards the evidence on the clinical side, *i.e.*, the

angular gyrus, there are now a fair number of cases, some with *post-mortems* in which loss of sight, not of retinal halves, but an amblyopic defect in one eye has been shown to depend on a lesion of this area on the opposite side to which the principal long sight exists, there being always a smaller loss in the eye of the same side also. (Ferrier, BRAIN, vol. iii., p. 456, and "Gowers," vol. ii., p. 19, and Sharkey, *Med. Chir. Trans.*, 1884, p. 265.) So many cases are now on record that Gowers' theory ("Manual," p. 19) of there being a "higher visual centre" in front of that for the half fields has been very generally accepted, and Ferrier's dictum as to this point being the centre for distinct vision ("Cerebral Localisation," p. 58), *i.e.*, from the macula lutea, has been shown to be most highly probable. I would therefore venture to admit that the macula lutea is represented in this area exclusively, chiefly of the opposite eye, and to a smaller extent for the same side as that of the lesion causing crossed amblyopia. But in this particular there is one fact somewhat difficult to account for, namely that in all these cases reported the clinical symptom has been a peripheral restriction of the field, and not a loss of central vision as one would have expected. The cases have mostly been so carefully reported that a mistake is scarcely possible, and on the other hand it is almost without doubt that the loss is one of central vision; it is difficult to reconcile the discrepancy, but the facts are so distinct that one must be contented with simply finding the explanation, and I can only suggest that as the representation is bilateral, and that therefore the opposite hemisphere also subserves this very area, *i.e.*, the fovea, it is that which is retained though much lessened.

A series of cases have been reported shewing that lesions of the angular gyrus of the left side cause word blindness, and the relation is now fairly established, as also is the fact that the affection is not of necessity accompanied by any affection of vision in its ordinary sense; if there is any one occupation *par excellence* requiring the use of clearest vision and therefore of the yellow spot, I should imagine it to be that of reading, so that this symptom following a lesion

of this area is scarcely to be wondered at, and its cortical limitation to the left side is only a further amplification of the general rule in that respect as regards all the other varieties of aphasia. As word blindness is not necessarily accompanied by visual loss, though it should accompany a right-sided amblyopia, theoretically, its explanation is probably that of a partial lesion or the partial and remaining effect of a lesion otherwise compensated for showing itself in a loss of those faculties latest acquired, visual ideation suffering more readily and more lastingly than simple visual perception.

The same may be said of the cases of mind blindness described as occurring clinically, for example, those of Chauffard (*Rev. Med.*, 1881, p. 440), and MacEwen (*British Medical Journal*, 1888, p. 306), and possibly of the similar condition described in the lower animals by Munk and Goltz. If the cell modifications which presumably occur as a result of a stimulus, *i.e.*, a perception, being, as it is reasonable to suppose they are, permanently impressed there or bearing an "impression" and so constituting in each a residual path of lessened resistance and thus in each sensory centre, the organic basis of the memory of its own sensory impressions one can understand, even without resorting to the cumbrous theory of a portion of the cortex being reserved for the storing up of visual images, the fact of the possibility of a lesion here causing inability to recognise words and even objects, the latter I should imagine being a relatively more severe form than the other.

It may be urged that a similar loss in other senses should be theoretically possible, *i.e.*, that a man could be mind deaf as well as word deaf and unable to understand sounds just as he is unable to interpret words, though he could give evidence (spontaneously only) of hearing them. Or even one might suppose either smell or touch to be similarly affected. But apart from the extreme difficulty of proving such defects, vision is in man undoubtedly the highest intellectual sense, and probably on that account its acquirements are the easiest lost or damaged. This may be stated in other words, that of all the senses in man, I should be

inclined to say—smell ranking next to it in this respect—it is the one least dependent on the others for association, that whereas the stimulus of, and *ipso facto*, the memory of the impression of hearing, touch, &c., are associated usually with those of others, that of vision is often quite solitary, and therefore if lost would have fewer aids to revivification, re-ideation and memory. A sound we hear is generally associated with the sight, perhaps too the tactile perception of its causal agent, a touch equally with vision, sometimes with hearing or even other sensations, while a visual impression may arise *per se* quite unaccompanied by any other sensory stimulus, and so be much more liable to loss of representation, having no other associated memory that it is possible to recall.

It may be here mentioned that a possible explanation of the angular gyrus being particularly liable to lesions and partial lesions lies in the fact of its being at the junction of the different blood supplies, the posterior and middle cerebral, and though usually being supplied by the latter, the former occasionally instead or in addition giving blood to it.

It would seem curious that no analogous condition to either word or mind blindness should have been ever described as resulting from occipital lesions, but I would venture to submit again that this coincides with the theory enunciated in this paper, the angular gyrus being peculiarly for distinct central vision as used when the object is to be and is most clearly defined, and *ipso facto* most clearly perceived, and a "visual impression" *sic* of it retained and rendered capable of future representation, while the occipital lobe is solely for general binocular vision; possibly every portion of this relatively large area being divided into functionally separated segments related to correlated separated portions of the retina and the general visual field, even without supposing these to be a separate portion of it required for the representation of colour vision; for it is well known that cases have been recorded of hemiopia for colours alone, there being now a good many recorded, and from this fact several authorities have considered a separate cortical centre for colour vision to be necessarily present.

The affection has been usually attributed to lesions of the occipital lobe. I am not aware of any case having been found due to disease of any part of the parietal lobe; indeed in the cases of amblyopia the colour fields are generally reduced in ratio with that for light, as is the ordinary rule in hemianopsia of cortical origin, and in chiasmatic (temporal hemiopia) the colour fields are equally lost. In optic nerve atrophy, as is well known, colour blindness is in nearly all cases well marked, and seems often at least to bear no relation to the loss of visual acuteness.

It may possibly be that there is a separate centre for colour vision in the cortex, and if so, colours not requiring fixed and clearest vision for their perception, it would presumably be in the occipital lobe. But if the physical perception of colours really depends for each one on the relative number of vibrations of light ether, it may also well be that the same part of the cortex as for light subserves this formation, and that a less severe lesion, *i.e.*, a relative one, could cause this symptom, it may possibly have some relation to the function of different layers of cortical cells. I am not aware if it has ever been shown to follow lesion of the tract or radiation which would, of course, under the theory of a separate area for its centre, be quite possible; also it seems that in most cases of "hemiachromatopsia," there is generally some affection of ordinary vision, and that as the loss for each colour is often so very different, and that it is possible for persistent blindness to one or more colours to remain as in the case quoted by Gowers ("Manual," vol. ii., p. 146), while the others may be imperfectly perceived, it would appear to be necessary, if there is a separate area for the centre for colour vision, that there should be one for each individually and separately. It is much more reasonable on these grounds to suppose the colour loss to be due to a lessening of function, which, if more pronounced would cause a loss for light.

I have purposely refrained from alluding to the result of experiments on animals other than monkeys, for in these former the brain is so unlike in general appearance that of the human subject, that it seems as though the clearest evidence for a corresponding nomenclature of parts is a

corresponding response to experimental lesion or irritation, and to argue from this backwards as to the seat of a function in similar named areas would, I think, be rather a *petitio principii*, but still speaking broadly and without going into any minutiæ, it seems as though even in these animals there was as in man, a distinct and separate representation for (1) clear vision and (2) for the correlated retinal half fields.

Bechterew (quoted by Ferrier "Cerebral Localisation," p. 65) found in dogs and cats two regions functionally differentiated in this way, one posterior related to the retinal halves, and another anterior to this in the middle of the second external convolution in relation with the opposite eye alone. It seems as though the same facts may be deduced from the very opposing results recorded by Dalton, Loeb, Goltz, Luciani, Tamburini and others, some stating that lesions of the visual areas in these animals caused crossed amblyopia, while others declared the visual loss to be a hemiopic one.

I cannot help thinking that the explanation is such as has been stated; and that it will be found on examination that the general scheme of visual representation in nature is for the whole field of the opposite eye most probably as in man, each eye being represented on both sides of the brain, though this is impossible to determine in animals; and that in those animals provided with binocular vision the homologue of an occipital lobe exists for correlated halves of each retina.

THE SENSORY SIDE OF APHASIA.[1]

BY E. A. SHAW, B.A., M.B., SURGEON, R.N.

THE consideration of this subject may best be commenced by a short sketch of its history, which is indissolubly welded with that of the localisation of the faculty of language as a whole in the brain cortex. Gall appears to have been the first to have placed the faculty of speech in the anterior lobes of the brain, regarding it as more or less in relationship with the frontal eminences of the skull, but it would seem that he so placed it from *a priori* reasoning, and more as a quasi-scientific speculation than as an induction from the consideration of an assemblage of ascertained facts. The first serious attempt to locate it by means of morbid anatomy seems to have been made by Bouillaud in 1825, who, in his "Traité de l'encéphalite," placed it in the anterior lobes of the cerebrum.

The next worker on the subject appears to have been Marc Dax who is stated to have published at Montpellier in 1836 an account of a "Lésion de la moitié gauche de l'encéphale, coïncidant avec l'oubli des signes de la pensée," in which he localized it in the left hemisphere near to the island of Reil. Bernard, however, in his work on "l'Aphasie" published in 1884, goes very deeply into the question of the validity of the claim of Marc Dax to be regarded as almost the pioneer in this field of pathology, and the evidence he adduces makes it very doubtful whether Dax's memoir, though doubtless it existed in 1836, was ever read publicly; it was apparently first published only on the 25th May, 1865; and was certainly all but forgotten till Broca's

[1] Communicated by the Director General of the Medical Department of the Navy.

famous and now historical paper "Sur le siège de la faculté du langage articulé avec deux observations d'aphémie," appeared in the *Bulletin de la Société Anatomique*, tome vi., August, 1861, and placed the faculty of language in the third left frontal convolution.

In the same year Charcot published in the *Proceedings of the Anatomical Society of Paris* a most interesting case (quoted at greater length in the list of cases) in which there was right hemiplegia and aphasia, and at the *post-mortem* the frontal part of the brain was found to be perfectly normal, but the first and second tempero-sphenoidal convolutions, island of Reil, extra-ventricular nucleus of the corpus striatum, and intra-ventricular nucleus in its posterior half had been destroyed by softening. Broca appears to have examined the case during life, and was certainly present at the *post-mortem*, which is described as having been conducted with the greatest possible care, and at which he admitted that this case was distinctly at variance with his hypothesis. Its true bearing we now easily see, but for some time it was regarded as an insuperable objection to Broca's hypothesis, and then later, as cases similar to Broca's multiplied, as one of those exceptions that prove the rule.

Montpellier seems to have been peculiarly connected with the subject of aphasia, for one of its professors, M. Lordat, presented a most striking instance of the disease, viz., entire inability to speak, due to loss of memory for words with complete word-deafness and word-blindness: he could spell but not read; in his own words "Je possédais complètement la partie interne du langage, je rien avais perdu que la partie externe."

Hughlings Jackson (*London Hospital Reports*, 1864) first brought into prominence the distinction between the use of emotional speech (oaths, interjections, &c.) and conceptional or propositional speech (*i.e.*, speech embodying some idea).

Marce (*Mem. de la Soc. de Biol.*, tome iii., 1865) was the first to maintain that the centres of co-ordination for spoken and written words are different and separated from each other.

Ogle (*St. George's Hospital Reports*, 1867) arrived at a similar conclusion from a consideration of cases in which agraphia and aphasia were separated; he also placed the two centres close together, and drew a distinction between "amnemonic aphasia" in which inability to translate ideas into symbols was due to forgetfulness of words, and "atactic aphasia" in which the defect was due to want of cöordinating power over the muscles of articulation; and further applied the same distinction to agraphia.

Jaccoud (*Leçons de clinique médicale*, 1874, page 65) supposed automatic speech to be presided over by a basal centre of sound situated underneath the corpora quadrigemina, but this was not much more than a speculation.

Charlton Bastian, according to a review by de Watteville in BRAIN for July, 1885, appears to have been the first to draw attention to the sensory nature of word-blindness and word-deafness in an article "On various forms of Loss of Speech in Cerebral Disease," which appeared in the *British and Foreign Medical Chirurgical Review* for 1869. Broadbent, in a remarkable paper on the "Central Mechanism of Speech and Thought" (*Medico-Chirurgical Transactions*, 1872), adopted the hypothesis of distinct perceptive centres; and Wernicke ("Der Aphasische Symptomen-Complex," Breslau, 1874) still further defined the localisation of an auditory word-centre and placed it in the first temporal convolution. And since this the subject has received much attention at the hands of Bastian, Broadbent, Kussmaul, Habershon, Wernicke, Kahler and Pick, Fritsch, Ross and Giampietro.

I now propose to quote a number of cases which I have collected from various sources, including one of my own. All these have been selected from the very extensive literature of the subject as illustrative of the sensory side of aphasia, and in addition as having a *post-mortem* record attached; they will be followed by a tabular statement of their main features, both clinical and pathological.

Case 1.—The first case of importance published after Broca's celebrated paper was the one by Charcot, which appeared in the *Proceedings of the Anatomical Society of Paris*, late in the year 1861. The patient was a woman of 47, who, consequent on a fit

of apoplexy, presented right hemiplegia and aphasia. Her intelligence was unaffected, and memory good, but her articulate language was reduced to the monosyllable "Ta" repeated several times over ("ta, ta, ta, ta") very rapidly and distinctly each time she tried to answer any question or to communicate her own ideas. The tongue was perfectly free and could be moved in any direction.

Post-mortem.—It was found that softening had destroyed the first and second temporo-sphenoidal convolutions, island of Reil, extra-ventricular nucleus of the corpus striatum and intra-ventricular nucleus in its posterior half; the optic thalamus was intact; the frontal convolutions presented no alteration either in volume, colour or consistence. The examination is described as having been conducted with the greatest care in the presence of MM. Cornil and Broca, the latter of whom, as has already been mentioned, admitted the case to be at variance with his hypothesis.

Case 2.—A case published by Bateman in his work on "Aphasia." Wm. Sainty, a waterman, aged 51, on Dec. 9, 1864, went into a tavern to order some beer, when he found himself totally unable to speak. There were no other paralytic symptoms; memory and intelligence were unimpaired. At the end of a fortnight he could speak a few words, but in March, 1865, he had a kind of fit, falling down and remaining for a few moments unconscious. He was then admitted into hospital. Intelligence unimpaired, no want of sequence in his thoughts, partial agraphia, can form one or two words but cannot write a complete sentence. Deglutition unaffected, movements of tongue unimpaired, but right half is slightly raised above left half and is more flabby, and when told to protrude it, he holds it out a long time as if from defect of memory, probably not remembering what he had done. Organs of special sense unimpaired. In May, there was a slight improvement more marked in writing than speaking. During 1866 he had fits every few weeks.

Re-admitted January 12, 1867. No diminution of intelligence or motor power, no paralysis. Understands all that is said, but has lost the memory of words so far as substantives are concerned. If asked to bring anything he brings the right article, but if he wishes to ask for anything himself, he usually asks for the wrong thing, afterwards correcting himself, saying that he knew it but could not recall its name, and on being asked was it this, that or the other thing, he invariably recognised the proper name for it.

April 24, 1867.—Sudden lower paraplegia and speechlessness, upper limbs unaffected. In May he was admitted into the Borough Asylum, quite imbecile and unmanageable.

July, 1868.—Though unable to articulate he gesticulates frightfully, endeavouring by language of signs to supply the loss of articulate language. He died at the end of the month.

Post-mortem.—Intense congestion of convex surface of hemispheres especially on left side, considerable opacity of arachnoid also on left side, a *little* flattening of convex surface of left anterior lobe. In middle of left posterior lobe was a softening about the size of an apricot; a similar condition existed at the same spot in the right hemisphere, but the softening was not so far advanced. The frontal convolutions were carefully examined, as were the central ganglia, pons and cerebellum, and all found healthy. These points were subsequently verified by microscopical examination.

Case 3.—Also reported by Bateman in the same work. Mr. N., a merchant, aged 51, consulted him in March, 1867. He was able to speak, but on being questioned got confused, saying he could not find words to express his symptoms; what answers he made were quite coherent, but given in the fewest possible words. He had lost the memory of many substantives and would call things by their wrong names, afterwards correcting himself.

Post-mortem.—At point of union of middle one-third with posterior one-third of convex surface of left hemisphere was an arachnoid cyst under which was a well circumscribed portion of softened cerebral tissue about the size and shape of an apricot. Anterior hemisphere was quite healthy.

Case 4.—Published by Broadbent in a paper on the "Central Mechanism of Speech and Thought," *Medico-Chirurgical Transactions*, vol. lv., 1872, p. 162.

An intelligent man of 59, after an acute cerebral attack lost completely the power to read printed or written words; he was also unable to recall the name of the most familiar object presented to him. He could, however, converse fluently, his vocabulary was large, and his words well-chosen and arranged, although he occasionally forgot the names of streets, persons and things. He could also write easily and correctly, both from dictation and spontaneously.

Post-mortem.—He died from an extensive hæmorrhage into the left temporal lobe with rupture into the lateral ventricle. Two foci of softening of older date were observed, one being situated in the temporo-sphenoidal lobe beneath the posterior end of the parallel sulcus, the other higher up underlying the angular gyrus, and between it and the point where the descending horn of the ventricle is given off.

Case 5.—Also published by Broadbent in the *Medico-Chirurgical Transactions* for 1878.

A man aged 60, who had previously been a good talker and a great reader, suffered, after a fit of some sort, from a peculiar affection of the speech, with paresis of the right side of the face but no hemiplegia. His speech was a mere inarticulate jargon. When asked a question he would make a brief reply as if he understood and answered; the modulation of the voice and the emphasis were perfectly natural, and corresponded with the facial expression and gestures, but as a rule there was not the least semblance to words in what he said. His replies were often so suitable in length and emphasis that it might have been supposed that he had comprehended the question. When, however, he was told to do anything it was seen that he did not understand the simplest phrase. He sat up in bed once or twice when required to do so, but as this was not made a test question, there would be other indications of what was wanted, and he was extremely ready in comprehending signs. When told to give his hand he invariably put out the tongue. A letter addressed to him at the hospital being handed to him, he took it, appeared to read the name and address and put it down again, not attempting to open it. A piece of paper having on it, "Give me your hand" was handed to him; he took it, held it so as to get a good light on it, then having apparently read it, laid it aside without giving his hand, though asked to do so by word of mouth as well as in writing.

Autopsy.—A large focus of softening was found in the left hemisphere limited to its posterior half. Part of the supra-marginal lobule was yellow in colour, shrunken in volume and soft. This condition extended upwards and backwards to within about half an inch of the longitudinal fissure just in front of the external parieto-occipital fissure, involving therefore the postero-parietal lobule. The morbid change implicated the angular gyrus and nearly reached the occipital lobe. The adjacent parts of the temporo-sphenoidal lobe, the posterior end of the infra-marginal and the parallel gyri were soft but not wasted or discoloured on the surface.

Case 6.—A case published by Kahler and Pick "Beitrag zur Lehre von der Localisation der Hirnfunctionen," *Prager Viertel Jahrschrift*, bd. cxli., 1879. There was well marked word-deafness, and *post-moriem* there was found a lesion situate in the first and second temporo-sphenoidal convolutions of the left side.

Case 7. — A similar case published by Fritsch, *Wiener Medizinische Presse*, 1880. Well marked word-deafness, and *post-*

mortem a lesion of first and second temporo-sphenoidal convolutions of left side.

Case 8.—Also a case of word-deafness published by Wehrnich, "Lehrbuch der Gehirnkrankheiten fur Aerzte und Studirende," Band ii., 1881. At the autopsy a focus of softening was found in the first and posterior part of the second temporo-sphenoidal convolutions of left side.

Case 9.—A case of severe amnesic aphasia reported by Kussmaul in his article on "Disturbances of Speech" in Ziemssen's Encyclopædia.

A stonemason of 55, in the clinic at Freibourg, presented a typical picture of loss of memory for spoken and written words without special complications, this manifesting itself mainly by a forgetfulness of substantives and the markedly disjointed manner in which he combined letters and syllables in spoken words; recognition and comprehension of words not being noticeably affected.

No note about vision save that there was much presbyopia.

Autopsy.—Two foci of yellowish-red circumscribed softening on left side; one 5.5 cm. × 1.1 to 1.8 cm. occupied the anterior position of the gyrus angularis; the other 2 cm. × 0.6 cm. had destroyed partially gyrus occipitalis ii. where it unites with gyrus temporalis ii.; the depth of these foci was only 4 mm.

Case 10.—A case reported by Broadbent "On a particular form of Amnesia; loss of nouns," *Medico-Chirurgical Transactions* for 1884.

A gentleman of 72, developed slight hemiplegia in September, 1878, and died in December, 1883. All this time he seldom used a noun-substantive, and when he did, it was wrongly used. He could not name an object at sight, could not repeat nouns after hearing them, though when told to say "1, 2, 3," did so promptly. He clearly understood all that was said to him, read newspapers and pointed out paragraphs relating to people he knew. His memory was good, but he could not answer a question in writing. There was well-marked right hemiplegia and anæsthesia.

Autopsy.—The right hemisphere was normal.

Left Hemisphere.—Posterior *outer* and two long posterior convolutions of the island of Reil had entirely disappeared. The angular gyrus round the margin of the Sylvian fissure and the super-marginal gyrus forming its upper margin posteriorly were undermined by an extensive area of degeneration continuous with that which has destroyed the posterior insular convolutions. The

auditory perceptive centre had entirely escaped. The mass of fibres running from behind forwards under posterior convolutions of island of Reil, converging from angular, supra and infra-marginal, parallel and other gyri to the two ascending parietals and posterior part of third frontal, had been destroyed.

Case 11.—A case reported by Osler in the *International Journal of Medical Science* for March, 1891.

J. W., aged 72, a Scotchman, by occupation a book-keeper. Inability to read a newspaper was the first sign; there was typical word-blindness. Philadelphia he spelt aloud "P r i n g r e k." He named objects held before him quite readily; there was no word-deafness at all. There was misplacing of words and sentences (paraphasia). In alluding to a wetting, he described it as "deliberate attacks of wet dress." There was right lateral homonymous hemianopsia, no paralysis. These conditions persisted over two months with gradual loss of muscular strength and mental power. For thirty-six hours before death there was paralysis of right arm and leg.

Autopsy.—Neurotic softening in the left hemisphere of the supra-marginal and lower part of the angular gyri, of the posterior part of the first and second temporal and of the two annectant convolutions uniting the first temporal to the parietal; there was complete transverse softening of the white matter between these convolutions externally and the lateral ventricle; the grey and white matter of the occipital lobe was uninvolved, but there was interruption of the fibres of the optic radiation. The anterior lobes and right side of hemispheres were healthy.

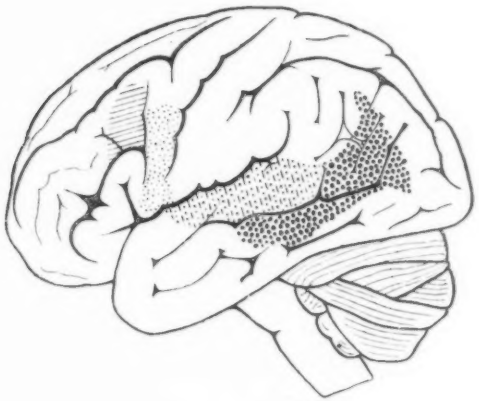
Case 12.—A case reported by myself in the *British Medical Journal* for February 27, 1892.

M. A. W., aged 72, widow, housekeeper, admitted into West Riding Asylum, January 30, 1890. She exhibited almost complete aphasia, agraphia and considerable dementia. She persistently repeated "Win yo? win yo?" (will you) and "man," and these were the few intelligible among many absolutely unintelligible utterances. She would frequently attempt to write, but would allow the pencil to fall from her fingers and go on with the motions of writing as though it was still there; she never succeeded in making a single recognisable letter. She was quite cheerful and accompanied her attempts at talking by gestures, the meaning of which was quite obscure, hence it could not be distinctly ascertained whether word-blindness as well as word-deafness was present. There was some paralysis of right side of face, not absolute;

distinct feebleness of right lower limb shown in walking but no dragging. Right upper limb was paralysed for fine movements, but was free from contractions and retained moderate grasping power.

February 26.—She could not apparently grasp the meaning of the most simple remark supplemented by gestures which would ordinarily make the meaning quite unequivocal. For months before her death in September, 1891, she made repeatedly most vigorous attempts to make herself understood, getting quite vehement at times but only succeeding in uttering unintelligible jargon, and often weeping at her consequent failure to make herself understood.

Post-mortem.—Diploë of skull cap softened. Dura mater a little thickened; there was a considerable quantity of fluid in subarachnoid space, especially on left side, where the visceral arachnoid was raised by it from surface of convolutions and pia mater in some places to extent of half an inch. On left side of brain there was considerable orange-red softening of superior and part of middle temporo-sphenoidal convolutions, and of the angular gyrus



and also of the convolution of the surface of the island of Reil. There was a less degree of softening at the junction of the second frontal with the ascending frontal, and the latter itself was affected with softening but to a slighter extent than any of the other diseased areas. On section the white matter between the claustrum and external cortex was seen to be involved also.

ANALYSIS OF CASES.

No. of Case.	*Prominent Clinical Features.*				*Main Pathological Lesions Affecting*							
1	Word-deafness	Speech reduced to "ta-ta-ta"			1st and 2nd temporo-sphenoidal convolutions						Island of Reil	
2			Loss of memory for substantives									Middle of left posterior lobe
3			Loss of memory for substantives									Junction of middle and posterior thirds of convex surface of left hemisphere
4				Word-blindness	Temporo-sphenoidal convolutions	Area underlying the angular gyrus						
5	Word-deafness	Speech jargon		Word-blindness	Temporo-sphenoidal	Angular gyrus	Supra-marginal	Postero-parietal				
6	Word-deafness				Temporo-sphenoidal I. and II.							
7	Word-deafness				Temporo-sphenoidal I. and II.							
8	Word-deafness				Temporo-sphenoidal I. and II.							
9		Propositional defect in speech	Loss of memory for substantives			Angular gyrus			Occipital II.			
10			Loss of memory for substantives			Angular gyrus	Supra-marginal			Commisural fibres from hind- to fore-brain	Island of Reil	
11			Propositional defect	Word-blindness	Temporal I. and II.	Angular gyrus	Supra-marginal					
12	Word-deafness	Speech jargon			Temporo-sphenoidal I. and II.	Angular gyrus					Island of Reil	

Proceeding now to a consideration of these cases, let us see what may be deduced from them. I am only too conscious that they are lamentably few in number for the purpose, the cause of this being the limitation of cases chosen to those with a *post-mortem* record attached, and in the most exhaustive possible examination of the literature of the subject, though I have met with some hundreds of reported cases of sensory aphasia, these twelve were the only ones I found with such a record.

Taking first those presenting the features of "word-deafness" we have six cases, Nos. 1, 5, 6, 7, 8, and 12, and each of these cases is associated with disease of the temporo-sphenoidal convolutions; in five of them the convolutions are distinctly specified as being the first and second, in the remaining one (case 5) it is simply stated that part of the temporo-sphenoidal lobule was softened. The association is obvious. On the other hand, we have two cases, Nos. 4 and 11, in which there was disease of this region without apparently any word-deafness at all. Of course it is possible that the lesion was not sufficiently severe to produce this effect in these two cases, and we may regard this as neutral evidence so far as the localisation of a cortical centre associated with word-deafness is concerned, for the series does not contain a single case exhibiting word-deafness in which there was not disease of the temporo-sphenoidal convolutions with the one doubtful exception of case 10, in which the patient was unable to repeat nouns after hearing them, and this Broadbent regards as a case not of word-deafness, but of loss of memory for substantives, of which the series I have collected presents several instances. Therefore, from the pathological standpoint we may say there is a distinct association between the first and second tempero-sphenoidal convolutions and the phenomenon of word-deafness; in other words, that in these convolutions is situated the "auditory perceptive centre." This is where Ferrier, from considerations of quite another nature, places it, and though the experimental researches of Schäfer and Sanger Brown as to the position of the auditory centre in monkeys do not support this conclusion, neither do they contradict it; they

are of a negative nature. Giampietro (*Ann. des Mal de l'Oreille, &c.*, March, 1893), I see, places what he terms the "ideo-phonic" centre in the first temporal (tempero-sphenoidal) convolution alone.

Taking next those cases presenting the features of "word-blindness," we have three cases, Nos. 4, 5, and 11, all associated with disease of the angular gyrus; it is true that in two of these (cases 5 and 11) there is also disease of the supra-marginal convolution, but, for reasons which I shall come to shortly, it is quite unlikely that the supra-marginal convolution has anything to do with word-blindness.

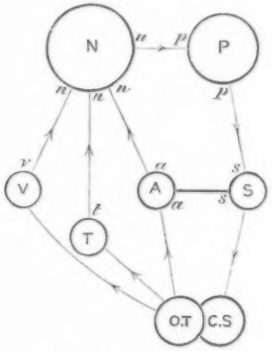
On the other hand, there are two cases (9 and 12) in which there was disease of the angular gyrus, but in which word-blindness is not recorded, but here, as in the consideration of word-deafness one must regard the evidence as negative, the word-blindness might have existed undetected, or the lesion of the angular gyrus might have been insufficient to have caused it, and as against it we have the fact that no case of word-blindness has been recorded where, *post-mortem*, the angular gyrus has been found free from disease. The location of a cortical centre for vision from considerations of experimental physiology is at present in a vague and unsatisfactory condition. Ferrier originally placed it in the angular gyrus, but later Munk showed that the occipital convolutions were concerned, and Schäfer went a step further and maintained that the angular gyrus had nothing to do with vision, and that the occipital gyri had everything to do with it, and that cases in which blindness was associated with disease of the angular gyrus, were simply due to the fact that the fibres connecting the optic tracts and the occipital convolutions, which run under the angular gyrus, had been destroyed. However, the three cases which have been adduced must be regarded I think, so far as they have any value at all, as supporting the view that the angular gyrus has a connection with what might be called "word-vision" rather than the occipital gyri, for in not one of them is the occipital lobe mentioned as being diseased; and also that there is a special centre for word-

vision, rather than that word-vision is the special activity of the general visual centre which is the first to be lost on its being affected by disease.

And now I come to the consideration of a variety of sensory aphasia, about which there has been much discussion, the very existence of which is denied by some writers, viewed as doubtful by others.

Broadbent is its most powerful advocate, and he names it "loss of nouns" or "loss of memory for substantives," regarding case 10 as a typical instance of it; and it may be noticed that in three other of the cases I have collected (Nos. 2, 3, and 9), it is mentioned as a particular feature.

Broadbent's diagram is as follows (BRAIN, vol. i., p. 493, 1878):—



V.—Visual centre (angular gyrus).
T.—Tactual ,, (uncinate gyrus).
A.—Auditory ,, (infra-marginal gyrus).

All sending tracts of fibres as indicated to "*N*" *the naming centre*. Here preceptions from V and T are combined into an idea, which can be symbolised by a name reaching N through A which has always been in the experience of the individual associated with the idea or object.

P.—Propositional centre in which a phrase is formed and mentally rehearsed.

S.—Motor speech centre.

Lesion of V or fibres V to N causes word-blindness.

,, A or fibres A to N causes word-deafness; and mistakes in words are not recognisable by patient, and A being the cortical guiding centre for S, lesion of fibres between A and S may result in mistakes in words which however, are recognisable by patient.

Lesion of S causes ataxic aphasia.

,, N would cause loss of memory of names or nouns.

,, P would leave patient unable to contruct a sentence, though retaining use of nouns.

Broadbent, in BRAIN, vol. i., 1878-79, p. 491 *et seq.*, puts forward the idea of a motor cell group—a "word group" situated probably in the corpus striatum— and postulates a distinct "naming" centre and also a "propositional" centre, in which phrases are formed and mentally rehearsed; the existence of this "naming centre" he further dwells on in an article in the *Medico-Chirurgical Transactions* for 1884, "On a Particular Form of Amnesia," regarding the case therein reported (above quoted as case No. 10) as due to the destruction of fibres connecting the "naming centre" with the motor-speech centre (third frontal convol.). Charlton Bastian (*British Medical Journal*, October 29, 1887) regards this propositional centre as merely a special activity of the general function of language, and the "loss of nouns" as a mere upsetting of the whole structure of language, and therein states that Ross holds similar views, though the latter in his work on "Nervous Diseases" ("Aphasia" in vol. ii.) mentions "Loss of Memory of Names or Nouns" as a subdivision of amnesic aphasia. Bastian further maintains (*Clinical Journal*, May 17, 1893) that "in ordinary silent thought the revival of words occurs primarily and perhaps mainly in the auditory centre."

There is, of course, in the very nature of the subject, any amount of room for speculation, and the thought naturally suggests itself, why should the revival of words in thought occur in the auditory rather than in the visual perceptive centre, to which it has been replied that the storing up of words in the memory is commenced and carried on for years as the child grows up, through the sense of hearing, and that from time immemorial in the majority of instances this has been the only way in which they have been so stored, the addition of words to the memory through the visual sight centre in reading, in both the history of the race and the individual, having been a recent and subordinate process assisted or possibly mainly carried out in most cases by the sense of hearing in the act of reading aloud, which is a not altogether improbable explanation why in the majority of educated people, new words and ideas are so much more easily grasped by means of lectures than by reading. Though the subject

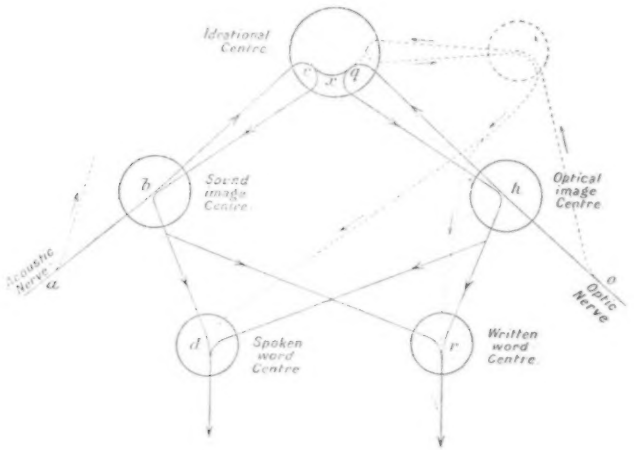
is, as yet, one mainly of conjecture, an examination of the schemes that have been suggested in explanation of the phenomena of voluntary speech may not be out of place.

There is no positive evidence of the existence of a naming centre; we have simply the occurrence of cases in which there is the one simple defect, the "loss of nouns"; and Broadbent says he has no evidence of a propositionary, as apart from a speech, centre, beyond the fact that cases are met with in which the patient cannot frame the simplest phrase and yet can repeat anything; these he would explain by assuming the propositional centre to be destroyed, the speech centre being intact. The converse, he says, would be conclusive, *i.e.*, the existence of cases in which the power of speech was lost, while the integrity of the propositional centre, as shown by the expression of ideas in writing, remained intact. I have come across three such cases, one reported by Trousseau (*Clinique Méd.*, tome ii.), another by Bouillaud (*Bull. de l'Acad. Méd.*, tome xxx.), and the third by Boinet (*Gaz. des Hopit.*, tome iii.); in all three patients the power of speech was lost, but their intelligence was unaffected, and all operations were perfectly conducted by means of writing. These certainly are cases for consideration which lend no little support to the view of the existence of a propositional centre, though why this centre should not be able at the same time to carry on the duties of the "naming" centre is a little difficult to see, and Kussmaul appears to be of this opinion, for in his scheme he regards them as forming one centre, which he terms the "ideational" centre.

Kussmaul's scheme is one which certainly cannot be accused of lacking completeness.

Comparing this scheme with Broadbent's, it will be seen that they have many points in common, the main points of difference being the fusion of Broadbent's "naming" and "propositionary" centres into one, the "ideational" centre of Kussmaul; the separation of Broadbent's speech centre into the two, one for spoken the other for written words, of Kussmaul; Broadbent, though he says (BRAIN, vol. i., 1878, p. 493) that the auditory centre is the cortical guiding centre for speech, does not represent the path from his proposi-

tionary centre to his speech centre as passing through the auditory centre; this omission is rectified in Kussmaul's schema. With regard to the path "o p d," which the latter regards as the one travelled in reading off aloud an uncom-

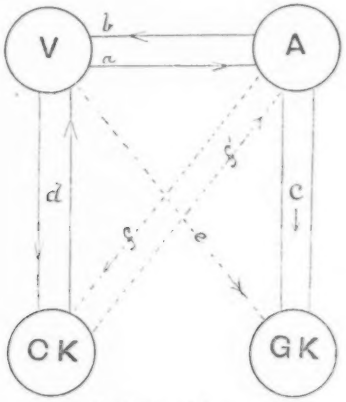


Kussmaul's Scheme.

a, b, c, b, d.—Collective acoustic motor track for spoken speech.
o, p, q, p. r.— „ optic „ „ written „
Punctated lines indicate paths from nerves of sense through other image centres to centre for conceptions (melodies, songs, gestures, &c.)
a, b, d.—Track for repetition of uncomprehended words.
o, p, r.— „ copying „ „ „
c, b, d.— „ conceptions expressed in spoken words.
q, p, r.— „ „ „ „ written „
c, x, q.— „ of connection between sound and text images, and renders feasible the change of spoken into written symbols through medium of thoughts.
a, b, r.—Track for writing down of an uncomprehended word that has been heard.
o, p, d.—Track for reading off aloud an uncomprehended written word.
a, b, c, b, r.—Writing down dictated words that are understood.
o, p, q, p, d.—Reading aloud written words that are understood.

prehended written word, I would suggest that considering the close relationship which has always existed between the sound image centre and the spoken word centre in the history both of the individual and the race; the much remoter con-

nection, and, if it exist, quite recently developed one as compared with the other, between the optical image centre and the spoken word centre; and also the fact that in a case of word-deafness, a patient, though he might make an attempt to read off aloud an uncomprehended word, would be quite unable to tell whether or not he was making a good or bad attempt; in short, regarding the auditory centre as the guid-



Bastian's Scheme.

V.—Visual word centre.
A.—Auditory ,, ,,
C K.—Cheiro-kinæsthetic word centre (writing).
G K.—Glosso-kinæsthetic word centre (speech).
d.—Visuo-kinæsthetic commissure.
b.—Audito-visual ,,
a.—Visuo-auditory ,,
c.—Audito-kinæsthetic ,,
f.—Audito-cheiro-kinæsthetic commissure } unimportant, stimuli only seem
f'.—Cheiro-kinæsthetic-auditory ,, } to pass along under "exceptional circumstances," which
e.—Visuo-glosso-kinæsthetic ,, } are not discussed.

ing centre for speech, I would suggest that the path "o p d" is hardly likely to be the correct one for the reading off aloud of an uncomprehended word, and that the more probable condition is the existence of a path from the optical image centre to the sound image centre, and that in the above instance the track is along this path and so to the spoken word centre;

and applying a similar line of argument, *mutatis mutandis* to the path "a b r" for the writing down of an uncomprehended word that has been heard, I should prefer to assume the existence of a path from auditory to visual centre and so to the written word centre rather than direct from auditory to written word centre.

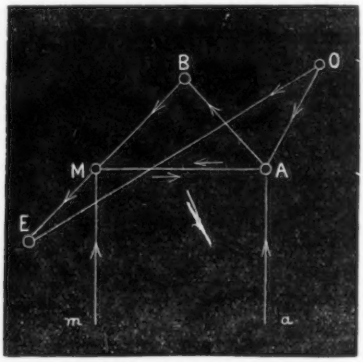
The existence of this cross-communication both ways between the auditory and visual centres is assumed by Charlton Bastian in his schema (*Brit. Med. Journal*, Oct. 29, 1887) which I shall now consider.

It will be seen that Bastian has no centre corresponding with either Broadbent's "naming" or "propositionary" centre or with Kussmaul's "ideational" centre; the functions of such possible centres he apparently relegates to the auditory centre; his own words are (*Clin. Journal*, May 17, 1893): "In ordinary silent thought the revival of words occurs primarily and perhaps mainly in the auditory centre. As I am speaking now, they come up quasi-spontaneously in the auditory centre"; and again (*Ibid.*): "In the same way, in the case of a person writing, the visual centre would be roused either primarily from the auditory centre, or, as in copying, by stronger impressions coming from without." Why the auditory centre should have been specially selected as the seat of that function of mind controlling thought does not seem quite clear, neither, assuming for a moment the truth of this hypothesis, does the course of the path by which thoughts are called up in the auditory centre by sensations of touch or taste, nor yet why thoughts thus called up should be developed in the auditory centre rather than elsewhere.

I now come to a consideration of Lichtheim's views, as propounded by him in a translation of his manuscript article by de Watteville in BRAIN, January, 1885; which appeared after being carefully revised by himself. Therein he puts forward several alternative schemes, the one receiving on the whole his preference being the one marked A on the next page.

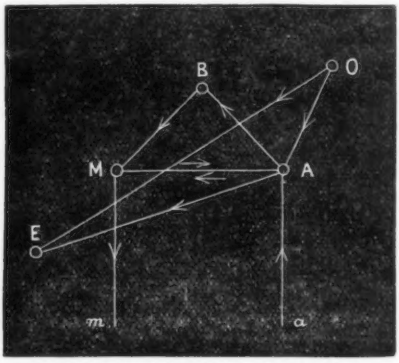
Having, however, a certain amount of doubt as to whether B M E be the path for volitional writing, he suggests as an alternative the diagram marked B.

It will be seen that in the latter diagram he does away with the direct communication from M to E, and makes his path for volitional writing B M A E.



(A)—*Lichtheim's Scheme.*

A.—Centre for auditory word impressions.
M.—Centre (kinæsthetic) spoken word impressions.
B.—Conceptional Centre.
O.—Visual word impressions.
E.—Written word impressions.
Reading aloud—O A M m. (The arrow should point downwards.)



(B)—*Lichtheim's Alternative Scheme.*

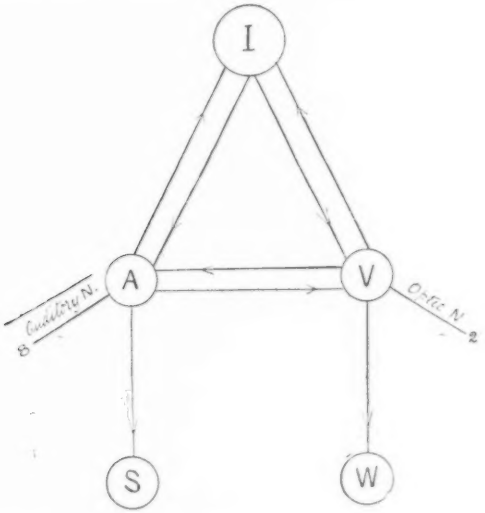
It is hardly to be supposed that Lichtheim would deny the existence of a path of communication from O, the sensorial visual centre, to B, the concept centre, though his diagram does

not recognise its existence, and presumably, confining one's self to the question of language alone, the path by which words in reading would reach B would be through O A B. Indeed, the Berne professor regards the auditory word impression centre as of primary importance; he says (*Ibid.*, p. 439) that damage to it involves the loss of understanding of written language (?) and as his diagrams show, the visual word impression centre he regards as communicating with the conceptional centre only through A. Now in the early stage of a previous education, this, I take it, undoubtedly is the case; the individual has to assist the process of grasping an idea from written symbols by reading aloud, and at a somewhat later stage, though the reading aloud is dispensed with, one sees the lips moving in correspondence with the words read; but in an advanced stage of education I think there is good reason for assuming the existence of a direct communication, recently established no doubt, between the visual word impression centre and the concept centre.

His views on the subject of word-blindness are somewhat at variance with those of other writers on the subject. He states (*Ibid.*, p. 484) that in cases of isolated word-blindness the patients can copy, and that in such cases we have not to do with a focal lesion in O, and that the break is in the tract O A, and thus again the visual word centre is subordinated to the auditory word centre. But surely it is possible by an injury to the visual word centre for word-blindness to be produced, and if A really occupies the position of all primary importance assigned to it by Lichtheim, how is a man who has lost the communication between it and O to copy words? The whole case for this assumption, it appears to me, depends on the statement on page 439, that damage to A involves loss of understanding of written language, which, though it may be the case in imperfectly educated persons, is not so, I venture to think, in their intellectual superiors, and hence a scheme providing for both possibilities becomes necessary.

I would now venture to suggest, not without considerable diffidence, the following as a scheme, including as far as possible the good points of its predecessors, at the same time minimising their defective ones, and also embodying the pre-

sent state of our knowledge of the subject as far as may be done. Of course it is bound to have defects and is largely speculative, but I venture to think it will serve as a working



I.—Ideational centre, naming, propositionary, can change spoken into written symbols "through medium of thoughts."
A.—Auditory centre, 1st and 2nd temporo-sphenoidal convolutions.
V.—Visual centre. Angular gyrus.
S.—Spoken word centre, 3rd left frontal convolution.
W.—Written word centre, 2nd left frontal convolution.
8, A, I, A, S.—Collective acoustic motor track for spoken speech.
2, V, I, V, W.—Collective optic motor track for written speech.
8, A, S.—Repetition of uncomprehended words.
2, V, W.—Copying of uncomprehended words.
I, A, S.—Conceptions expressed in spoken words.
I, V, W.—Conceptions expressed in written words.
8, A, V, W.—Writing down of an uncomprehended word that has been heard.
2, V, A, S.—Reading off aloud an uncomprehended written word.
8, A, I, V, W.—Writing down dictated words that are understood.
2, V, I, A, S.—Reading aloud written words that are understood.
Centres of perception for touch, taste, smell, &c., have been left out for sake of clearness.

hypothesis for the explanation of by far the great majority of cases of speech defect.

I have applied it to a large number of the published cases of speech defect of the kind under discussion, and I have in

every one yet tried found it capable of affording an explanation, speculative and theoretical though it may be, of all the clinical phenomena recorded.

I now come to a consideration of the nomenclature of this subject.

Ogle gave us the terms "amnemonic aphasia" and "amnemonic agraphia"; Ross speaks of amnesic aphasia, with subdivisions "word-blindness," "word-deafness," and "loss of substantives," as do many others: Broadbent, Bastian, Kussmaul, Lichtheim, &c. Kussmaul speaks of "simple aphasia of recollection." For speech-deafness Lichtheim proposes the word "logokophosis." It is a generally accepted theory that the language of medicine should be as cosmopolitan as possible, and with this object in view, the tendency is to fall back on the Greek language when attempting to coin words to express newly-discovered conditions. I would therefore venture to suggest that the terms "aphasia" and "aphemia" be restricted to defects in the processes of outgoing language of cerebral and bulbar origin respectively; that amnesia be used to mean Kussmaul's "simple aphasia of recollection."

I would suggest the word "aphemæsthesia" to express the general condition of failure of word perception, defect in ingoing language; and for inability to comprehend written symbols (word-blindness), *alexia*, a word used by both Kussmaul and Lichtheim in this sense. For inability to comprehend spoken language (word-deafness); instead of Lichtheim's logokophosis, I would suggest "kophemia," a shorter word whose Greek equivalents κωφος deaf, and φήμη speech, mean precisely the same thing.

Kussmaul's "*paralexia*," in which incorrect words are uttered, is now practically superseded by "*paraphasia*," and rightly, for λεξις means a written, not a spoken word, and the similar defect in writing is equally well expressed by "paragraphia," also a word which may be regarded as now fairly generally accepted.

In conclusion, I would urge that in the recording of these cases, the clinical phenomena should be investigated by means of some such schema as that published by Bastian in

his "Paralysis, cerebral, bulbar and spinal," and that the site of any lesion discovered *post-mortem* should be accurately recorded on one of the various brain charts now published for the purpose, and that when cases are published this should appear also; for it is only by the greatest possible accuracy in such details that contradictions in apparently ascertained facts are to be avoided, and a mass of accurate details accumulated, from which the factors governing these phenomena may be safely deduced.

THE "PILOMOTOR" OR "GOOSE-SKIN" REFLEX.

BY JAMES MACKENZIE, M.D.,
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In the careful examination of the skin of patients one frequently comes across a number of phenomena that are extremely difficult to understand. By accurate and minute observation of every detail, light is gradually shed upon the problems involved. They become all the clearer when these phenomena occur in definite areas which one can associate with the distribution of the nerve or blood vessels. When I first found out the occurrence of hyper-æsthesia in visceral disease, it appeared that there was a distinct connection between these areas and the distribution of the spinal nerves. These subjective symptoms sometimes bore a distinct relation in their distribution to those objectively presented in the eruption of herpes zoster. Similar data have been worked up by Dr. Head, into an elaborate delimitation of the fields of cutaneous distribution of the nerves from each spinal segment over the trunk and limbs. The deficiencies in one method of examination were frequently made good by the symptoms present in the other. While the hyper-æsthesia depends for its interpretation on the reliability of the patient, the occurrence of the eruption in herpes zoster, so far as it goes, affords striking and indubitable evidence independent of his sensations. It is very commonly found that patients are extremely inaccurate in regard to their sensations; and one has to be constantly on guard, not

only against the patient's misleading statements, but against one's own preconceived ideas. On the other hand, the eruption of herpes zoster is often unreliable on account of the imperfect manner in which one area is affected by the eruption, and also because we can never be certain when an attack of herpes is limited to one nerve root or one spinal segment,—according as we view the situation of the disease. For this reason, studying the subject of the cutaneous distribution of the spinal nerves from the purely clinical aspect, we are left in a somewhat indeterminate position. It behoves us therefore to be on the outlook for any other method that can help to strengthen our position. The results of injuries and lesions of the cord, while affording much valuable evidence, yet often fail us when we desire accurate and minute substantiation, on account of the impossibility of determining the exact limits of the effect of any lesion upon the cord. There are, however, several other phenomena which, if properly understood, can still throw light upon obscure points and substantiate doubtful results obtained in other ways.

The most striking of these is the occurrence of that condition commonly known as goose-flesh or goose-skin (*cutis anserina*). To me it is surprising that a phenomenon so common, and yet so instructive in its manifestations, should have been so universally ignored. All the text books on general medicine, skin diseases and physiology, as well as the larger encyclopædias, either contain no references whatever to this subject, or dismiss it with the briefest notice. Within recent years, two very interesting and instructive articles have been published by Langley and Sherrington,[1] based upon experimental researches, which shed much light on the relationship between the nerves, spinal and sympathetic, and the muscles of the hairs. What goose-skin is does not seem clear. In the New Sydenham Society's Lexicon *cutis anserina* is defined as "a term for that condition of the skin produced by cold and other causes in

[1] Drs. Langley, J. N., and Sherrington, C. S., "On Pilomotor Nerves," *The Journal of Physiology*, vol. xii., p. 278, 1891; also Langley, J. N., "The arrangement of the Sympathetic Nervous System based chiefly on observations upon Pilomotor Nerves," *ibid.*, vol. xv., p. 176, 1893.

which the papillæ become rigid and erect, resembling the skin of a plucked goose: goose-skin." According to the same authority goose-skin and goose-flesh are terms applied "to the condition of the skin called *horripilatio*, from its likeness to the skin of a plucked goose." In the same lexicon horripilation is defined as follows: (Latin, *horripilatio*, a bristling of the hair—from *horreo*, to stand on end, *pilus*, a hair . . . .) A sensation of creeping in the skin or as if each hair was stiff and erect in different parts of the body, producing *cutis anserina*." In Billings' "National Medical Dictionary" *cutis anserina* is "goose-skin; condition produced by contraction of pilar muscles over a large area," while horripilation is "shivering, with *cutis anserina*, with bristling of the hair."

These various definitions are too vague, and they scarcely embrace the full significance of the phenomenon. What all are agreed upon is that the symptom is due to a contraction of the pilar muscle with which each hair follicle is supplied. The symptom, however, need not, and frequently does not, produce a visible erection of the hair; but it usually does cause a projection of the glands or follicles above the skin, giving rise to the peculiar pimply condition that we associate with goose-skin. It also gives rise to that peculiar sensation which is associated with the production of goose-skin. This sensation is peculiar to itself, and to what it is entirely due I am not certain. The mere traction and shrinking of the skin does not appear to be its sole cause, because I find it present in areas where there is no evident goose-skin produced; and it even extends to areas where the hair follicles do not exist. Again, the goose-skin may be present when no such sensation exists. So far as my inquiry into the subject has extended, I find that I am far from having exhausted the information which is to be obtained from the study of this symptom. While it is capable of production in nearly all people, yet it varies individually, both as to extent of distribution and degree of excitability, and even in the same individual at different times. But its extent and limitation offer valuable and unique opportunities for the study of the distribution of the

nerve supply, in a manner which no other clinical phenomenon, with which I am acquainted, does so clearly. Thus it reveals certain definite boundaries in the distribution of the spinal nerves considered according to the distribution of each spinal segment. It also shows in suitable cases, the distribution of the nerve supply, considered according to the disposition of the peripheral nerve in a longitudinal or vertical direction, on the dorsal, lateral, and ventral surfaces of the body. It also brings out the dissemination of nervous impulses, in cases of visceral disease. Numerous as my observations have been, they are as yet far from complete; and I am unable to deal exhaustively with the information to be got from the study of this symptom. My object here is to call attention to a valuable symptom, affording a new method of investigation of a subject which has been recently discussed in these pages; I refer to the distribution of cutaneous nerves, and the sensory symptoms associated with visceral disease.

Every one, I suppose, is conscious in their own person of the production of this symptom over large areas of the skin. The sudden exposure of the warm body to the cold air, produces it most extensively. Under certain conditions, apparently consistent with perfect health, the symptom can be produced reflexly by stimulating the skin. The appearances when resulting from such reflex stimulation are either limited to the immediate neighbourhood of the part stimulated, or spread in a diffuse but still definite manner. Thus in stroking the skin firmly with a round, blunt, or broad-pointed instrument of one-quarter to half-an-inch in breadth, the goose-skin appears in the immediate tract of the part stroked and usually extends for some millimetres beyond on either side. (I am speaking now of the skin of the chest and abdomen; other parts do not usually manifest the symptom so readily.) From the area stimulated, the goose-skin may extend over a large tract of skin, but it does so in a very definite manner, the direction varying somewhat in different individuals. Usually it takes place along the areas of distribution of nerves, whose central connections are in close contiguity to those of the nerves supplying the

area stimulated, thus manifesting itself on successive fields of skin along a very definite path. When the stimulus is applied to the skin, one can observe each successive tract of skin becoming involved, although it occurs usually with such rapidity that one can scarcely determine the exact limitation at a given moment. In rare cases, the extension is so slow that one can follow it with fair certainty. In the observations I have made on the limited distribution, the methods of stimulation have been usually by stroking the skin with a blunt pencil-case, or by the finger covered with flannel or rough linen. At other times the whole trunk would be gently and equally rubbed with a towel while the goose-skin would appear only in very well-defined areas. I have said that the sudden exposure of the body to the cold atmosphere suffices to produce goose-skin, but it seems as if the slight friction produced in lifting the bed-clothes from the skin, is sufficient to set up a very wide-spread horripilation. I have tried to stimulate with cold, dry and wet—with pieces of ice direct, in an ice bag, and in test tubes—but without result.

So far as my observations go, I have found the goose-skin extend over the whole body, except the ears, hands, and wrists, feet and ankles. In one patient, the raising of the bed-clothes produced it over the whole body with the above exceptions, and also with the exception of the neck and upper part of the chest—an exception of some importance to be dealt with presently.

After the junction of the posterior and anterior roots of a spinal nerve, the nerve divides practically into three divisions which respectively supply the posterior, lateral, and anterior surfaces of the trunk. Among the limb plexuses certain modifications arise, but as my results do not yet permit me to discuss the distribution of the nerves in the limbs from a consideration of the pilomotor nerves, I shall not particularly refer to the extremities in relation to their nerve supply from this point of view. In figs. 5 and 6 of Ross's paper[1] the areas supplied by these divisions of the nerves are mapped out, and

[1] Ross, J. "On the Segmental Distribution of Sensory Disorders," BRAIN, 1888, p. 344.

agree fairly well with the areas marked out in fig. 1. Voigt[1] had indeed long ago demonstrated somewhat similar areas.

The reason I draw attention to this method of studying the distribution of the spinal nerves is because I have found occasionally that stimulation of a limited area of the skin will produce goose-skin, that spreads and fills exactly one or other of these areas. I have under observation two cases in which the goose-skin can occasionally be made to arise only in the area of the distribution of the anterior division, in the one case, and in the area of the distribution of the lateral division, in the other; and in neither case can it be produced in any of the other areas.

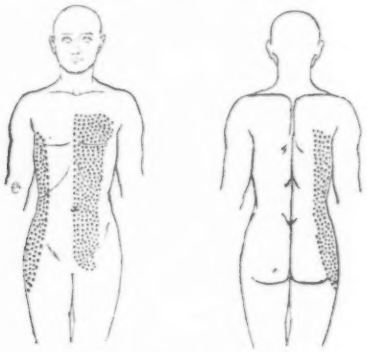

FIG. 1.

On the left side the dotted area represents the distribution of the goose-skin in the region supplied by the anterior divisions of the spinal nerves as in Case 1. On the right side the limits of the distribution of the goose-skin, back and front in the region supplied by the lateral divisions of the spinal nerve, are shown as in Case 2.

Case 1.—Male, aged 30. When the clothes are removed and he lies on his back, the temperature of the room being about 65°, stimulation by stroking the skin transversely in any part of the anterior surface of the left side of the abdomen, produces a typical goose-skin in a quick wave-like manner over the left side of the chest and abdomen. This area is limited *laterally* by a line

[1] Voigt, C. A., "Beiträge zur Dermato-neurologie; nebst der Beschreibung eines Systems neu entdeckter Linien an der Oberfläche des menschlichen Körpers," *Wien*, 1864.

drawn from the anterior part of the axilla to immediately internal to the anterior superior iliac spine, *internally* by the middle line, *superiorly* by the second rib, and *inferiorly* it fades away into the groin. Stimulation of any other part of the chest or abdomen produces no goose-skin. This area is extremely well-defined, and no difficulty is found in noting its extent. When this patient was stripped for examination in the out-patients' room, the goose-skin was much more universal. But it frequently happens that the areas most responsive to stimulation, vary from time to time.

Case 2.—Male, aged 29, complains of pain in his back, the result of a strain five years ago. When any portion of the skin of the chest or abdomen is stroked, the goose-skin spreads wave-like over the following area; it is limited *in front* by a line in the exact situation of that which bounded laterally the goose-skin in the preceding case, and *posteriorly* the limitation is equally abrupt in a line drawn through the angle of the scapula to behind the posterior crest of the ilium. *Superiorly* the goose-skin is lost in the axilla while *inferiorly* it descends over the hip, embracing the skin over the great trochanter (fig. 1).

Nothing could be more striking than the manner in which these areas were marked off. So distinct was the limitation of the goose-skin that one can but attribute it to some central cause. I incline to the view that the stimulation of the skin radiates along the paths that give rise to least resistance; and that in such cases as these, the central connections of the cutaneous nerves appear to be more easily excited longitudinally than transversely. That is to say, the excitation passes with more facility from portions of nerve roots supplying contiguous areas in the anterior or lateral aspect of the skin, as the case may be, rather than spread over the whole field of supply of one nerve. This view is strengthened by the consideration of what happens in the hyper-æsthesia in visceral diseases to which I shall presently refer. More commonly stimulation of a limited area causes the goose-skin to arise and extend by advancing over the whole half of the circumference of the trunk, although even here in its advance it sometimes shows a tendency to affect first, say, the anterior area for a limited extent, next the lateral, then the posterior, and then again a limited extent of anterior area and so on. This, however,

occurs so rapidly, that it can occasionally be detected only on its being specially looked for. The usefulness of the goose-skin reflex as a feature for observation is demonstrated in studying the manner in which it is limited, its boundaries presumably coinciding with the areas of distribution of definite nerves. Thus it very frequently happens that, in stimulation of one side of the chest or the abdomen, the goose-skin limits itself to one half of the body, ascending on the chest to a certain extent, then, instead of extending over the clavicles, passing down the arm in a remarkable and instructive manner. But not only does the goose-skin extend in this way, giving information by the limits of its distribution over a large area, but occasionally localized stimulation will produce the phenomenon in distant areas, which we know to be in anatomical relation. Thus stimulation of the upper part of the chest will produce the goose-skin over definite tracts in the arm; again, stimulation of the arm may sometimes produce it in certain definite areas on the chest, front and back. The following case first showed me the importance of the goose-skin as an object for study, and reveals features of such interest, that I quote it in full.

Case 3. — Female, aged 20, admitted into the Victoria Hospital, complains of loss of power in the legs. The patient got wet through three months ago, and her legs felt stiff the following day. Since then her power of walking has gradually become more impaired till now she cannot stand. On careful enquiry, she admits that there was weakness and a feeling of numbness in the left foot and leg, before the date on which she says she got wet. She also admits having had pain in the lower part of the spine. The patient is pale and fairly well nourished. She lies in bed with the legs bent at the knees, the right generally lying upon the left. She cannot voluntarily move the toes and ankle, but can, to a limited extent, move the leg at the knee and hip joints, but this she does stiffly. When forcibly bent, the knee offers considerable resistance, and gives rise to the clasp-knife phenomenon. The knee-jerks are exaggerated on the right side, normal on the left. Ankle clonus is readily developed in both legs. The plantar reflexes are greatly exaggerated, and the lower abdominal reflexes are absent. She has very little power over the bladder and rectum. Over both legs the sensation to

touch and pain is impaired and slightly perverted as high as the lower limit of the goose-skin in fig. 2, but there is no distinct border to be made out. The difference between heat and cold cannot be made out in the legs. The muscular sense is impaired. Over the lower third of the sacrum there is a diffuse swelling in the subcutaneous tissues, and the bone here is thickened with an indentation in the spine. The eleventh dorsal vertebra is tender to pressure. Goose-skin appears in certain areas on stimulating the skin. Thus, in testing for the sense of touch on the left side, by lightly stroking with a pin-head from the groin upwards along the abdomen, when the skin midway between the symphysis pubis and umbilicus was reached, the goose-flesh appeared over the abdomen at the part stimulated, and quickly spread in a wave-like manner up the chest, and into the left arm, over an area like that dotted in fig. 2. The lower limit extended irregularly round

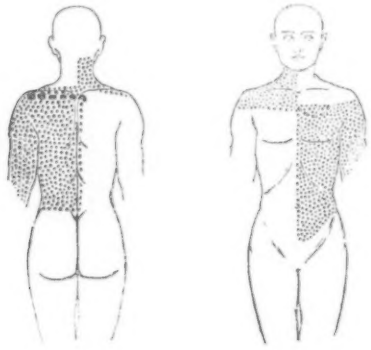

FIG. 2.

The dotted area on the left side represents the distribution of the goose-skin in Case 3. The portions above the interrupted lines in front and behind are those that were produced on stimulating the inner surface of the left upper arm. On the right side the dotted area represents the goose-skin in the distribution of the cervical plexus as in Case 5.

to the second lumbar vertebra. The lower margin was abruptly limited, and before it could disappear it was marked by a dermographic pencil presenting the same peculiar limitation as in fig. 2. The upper limit presented an interesting variation. As one watched the goose-skin ascend, it reached the level of the nipple, coursed round the upper border of the axilla, and descended down the inner surface of the arm for a short extent; then it reappeared

on the chest over an area extending from the level of the nipple to the first rib, at the same time descending further down the inside of the arm, and appearing on the back and outer side of the arm, not affecting the forearm nor ascending into the shoulder. If after it had subsided, we repeated the stimulation and observed the progress in the back, we noticed that when the goose-skin reached the level of the second dorsal vertebra, from which the upper limit extended outwards in a straight line, the goose-skin appeared next on the arm, then suddenly reappeared as a band above the previous limit, as high as the seventh cervical vertebra, while it extended lower on the arm and appeared on its outer and posterior aspect.

As it was evident that the goose-skin followed the distribution of the spinal nerves, numerous observations were made of the results of localized stimulation. It was not always easy to make out what the result was, except in one instance, which was frequently verified. If the inner surface of the upper arm was stroked (at various points which it was difficult to localize accurately) there appeared areas of goose-skin on the chest and the back, in the places situated above the interrupted lines drawn in fig. 2, representing presumably the distribution of the second dorsal nerve. Sometimes on the back and front of the chest, on stimulation of the anterior surface of the arm, the goose-skin appeared on the chest in a rounded patch over the second, third, and fourth costal cartilages, next round the nipple, then higher up, from the outer side of the breast to the anterior fold of the axilla; leaving an unaffected area above the nipple of about two inches in breadth. Stimulation of the inner surface of the arm would then produce the goose-skin in this unaffected area, followed by the extension over the whole half of the chest as in fig. 2. Stimulation of the outer aspect of the arm produced no goose-skin on the chest. These phenomena could be elicited also on the right side of the chest, except that the goose-skin never appeared so high on the outer aspect of the arm as it did on the left.

I was inclined at first to consider this excitability of the pilomotor reflex as dependent on the lesion in the cord; but that this is not so is evident from the study of a number of other cases with presumably healthy centres, of which the following is a most noteworthy example:—

Case 4.—Male, aged 35. Admitted to the Victoria Hospital on August 25, 1893, with a compound comminuted fracture of

the lower third of the femur. On October 25 it was found that there was no union, and the leg was encased in plaster of Paris. The patient is a man of ruddy countenance, well-nourished, and of powerful muscular development. Except for the fracture, he is in perfect health. In him the goose-skin is extremely active on first exposure of the skin to the air. After it has subsided, stimulation of the skin on the outer surface of the abdomen readily excites it, spreading rapidly over the left side of the chest and down the inner side of the arm and ulnar border of the forearm to the wrist. It leaves unaffected the radial border of the arm, but spreads over the outer and back part of the upper arm. The limits of the eruption are shown in fig. 3. On other occasions I

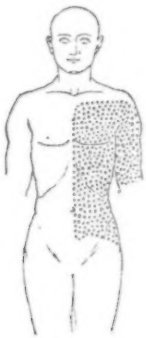

FIG. 3.

Distribution of goose-skin on stimulating any portion of the chest or abdomen on the left side. Case 4.

found that stimulation of the skin of the chest as low as the epigastrium on the front and side, produced the goose-skin over a limited area on the inner side of the arm, but not on the chest. This was repeatedly verified. There were some sensory phenomena produced, to which I shall refer later.

One very striking feature in these two cases, is the limitation of the area affected by the goose-skin. In the meantime I will only deal with the upper limit on the chest which is practically the same not only in both these cases, but also in a number of others I have observed.

It is evident, I think, that we have here the demarcation of the upper limit of the distribution of the dorsal nerves in

the chest, the area above this limit being supplied by the cutaneous branches of the cervical plexus. It happens, not infrequently, that one meets with cases in which the goose-skin is manifested only in the neck and upper part of the chest. The following is a fair specimen of this class:—

Case 5.—Female, aged 31; examined October 13, 1893, was seized yesterday with shivering and sore throat. The tonsils are slightly swollen, and inflamed with pus exuding from numerous follicles. As there was a case of scarlet fever in the house I exposed the chest to see if there was any rash, when I was struck with a well-marked appearance of the goose-skin that arose all over the neck and upper part of the chest. It was limited above to the hair-roots and under the ear and chin, while below it descended in front as low as the level of the second costal cartilage at the sternum, whence it extended outwards in a straight line reaching the level of the third rib in the nipple line, to the shoulder. Behind, the inferior boundary extended along the level of the first dorsal vertebra (see the dotted area on the right side of fig. 2). The goose-skin was much more easily excited on the *right* side than on the left; and the *right* tonsil was the more severely affected. After a few days the throat condition improved, but the goose-skin could still be obtained, though not so readily as before. A fortnight later the throat was still a little painful, and a faint goose-skin could still be perceived in response to stimulation. During the whole time the patient was under observation, it never tended to spread to any other region.

Before entering more minutely into the question how far the goose-skin reflex assists in determining the fields of cutaneous nerve-distribution, there are a few points which require elucidation, before we can confidently employ this phenomenon for such a purpose. At present I will do little more than indicate two of these points, namely, the question of overlapping, and the relation of the goose-skin to areas of skin exhibiting certain sensory disorders.

In regard to the question of overlapping, the study of this subject offers a very conclusive answer, so far as it concerns the pilomotor nerves. When the goose-skin is limited to one side, two conditions are present in regard to its relation to the middle line. If it be well-marked, it distinctly crosses the middle line and affects the opposite side to an

appreciable extent. This varies not only in different individuals, but in different places on the same individual. Thus in Case 3, in the epigastrium the goose-skin extended irregularly for one inch to the right of the middle line, when it was well-marked in the left half of the body. Above and below the epigastrium it extended across from one-quarter to half-an-inch. The method employed to demonstrate this with certainty, was to induce the goose skin in the left half of the body, then quickly to mark its boundary by the dermographic pencil. The right side was next stimulated, and the limits of the goose-skin thus produced mapped out in the same manner. It was found that a narrow strip of varying breadth down the centre of the body was affected, when the phenomenon appeared on either side. This overlapping could also be inferred from the further fact that, where the goose-skin on one side was but scantily present, it never quite approached the middle line; so that on stimulating one side, then the other, there was a narrow strip down the middle of the body in which the goose-skin did not appear. This was usually most evident on the back, where the goose-skin does not always manifest itself so distinctly as it does on the anterior aspect of the trunk. On account of this overlapping, the exact fields cannot be marked out on the skin of the body, where neighbouring areas of skin are supplied by neighbouring nerve roots. But when neighbouring areas of skin are supplied by nerve roots whose spinal origins are wide apart, then a very good opportunity is afforded for this method of studying the question of overlap. Thus the skin in the upper part of the chest is supplied by the branches of the cervical plexus, being conterminous with the area supplied by the second and third dorsal nerves—the intervening nerves supplying the arm. I have pointed out the striking manner in which the wave-like production of the goose-skin passes from the chest into the arm, leaving the upper part free. Now it so happens that this limit of distribution is very common and can be readily mapped out. On the other hand, it is not uncommon to find the goose-skin limited to the distribution of the cervical plexus. While, therefore, I have not been able to demonstrate these indepen-

dent areas in one individual, I have made careful observations of the exact limits in a number of people who presented either the one or the other localization. In these I found a remarkable agreement. In front the upper limit, where the chest was affected, extended usually to the level of the first costal cartilage and from thence outward to the top of the shoulder (figs. 2 and 3). On the other hand, where the area supplied by the cervical plexus was carefully marked out, it descended as low as the level of second costal cartilage and ran out transversely to the shoulder a little below the acromial process (fig. 2). Similarly behind, the upper limit of the goose-skin, when the chest was affected, ascended as high as the seventh cervical vertebra (fig. 2), while the lower limit reached as low as the first dorsal vertebra (fig. 2). These limits were not very abrupt, and at the margins the goose-skin faded away; but I have marked the limits where it could be demonstrated to exist without any doubt.

I have endeavoured to find out if there be any relationship between areas of disordered sensation and those exhibiting the pilomotor reflex. In Case 3, where the area of anæsthesia affected the legs and the lower part of the trunk, there certainly does appear to be a distinct connection, inasmuch as the reflex does not extend much beyond the limits of the healthy skin. But while the area invaded by the goose-skin is very distinctly limited here, that of the anæsthesia is not so distinct, there being a border of slight but varying breadth where it is difficult to determine whether the sensation is normal or otherwise. This border follows in its general contour the line round to the spine, indicating the lower limit of the goose-skin area. The latter never spreads into the parts where the anæsthesia is undoubted and permanent. From my investigations into the relationship of the pilomotor reflex with the hyper-æsthesia and pain of visceral disease, no definite conclusion can so far be drawn; but certain phenomena would lead one to expect that, in some cases at least, very instructive results may yet be obtained. Thus in a case of aortic disease, with well-marked attacks of angina pectoris in the left chest and arm, the hyperæsthesia affects the front of the chest from the level of the

second rib to the epigastrium. Behind the anterior axillary line the hyper-æsthesia becomes less distinct, its lower border slanting upwards to the spinal column about the level of the the third dorsal vertebra. The hyper-æsthesia also extends down the inner surface of the upper arm, ulnar border of fore-arm, to the little finger. The pilomotor reflex, when induced by gently rubbing the abdomen and chest of both sides, appears only on the left side. Its upper limit is situated as high as the second costal cartilage, and extends thence to the inner side of the upper arm, on which it descends as low as the elbow. But the upper limit of the goose-skin on the chest does not correspond exactly to the area mapped out by the hyper-æsthesia, inasmuch as the latter extends outwards to the axilla in an almost straight line from the second costal cartilage, while the upper limit of the goose-skin area curves down to below the third rib in the nipple line, rising again to the same height as the hyper-æsthesia in the anterior axillary fold. Below the goose-skin extends down the abdomen, fading away in the thighs in a manner too indefinite to be mapped out with certainty. No cutaneous hyper-æsthesia exists, and no goose-skin can be induced, in the area supplied by the cervical plexus.

In a case where there were a number of somewhat obscure symptoms which at first I thought were probably symptomatic of erythromelalgia, there were a number of patches of hyper-æsthesia following certain definite nerve tracts in the trunk and limbs, which it is not necessary to my purpose to give here in detail. Stimulation of the skin produced the goose-skin on the chest and abdomen, limited above as in figs. 2 and 3, and this was also the upper limit of an area of hyper-æsthesia that affected the chest. But the point to which I desire to draw attention here, is that when I stroked the skin with a blunt pencil, beginning on the side of the neck, downwards over the middle of the clavicles and down the chest to the level of the nipple, there appeared in the tract of the pencil a streak of goose-skin beginning abruptly at the line previously marked as the upper limit to which the goose-skin and hyper-æsthesia extended, that is, at the level of the second rib.

Above the second rib no signs of the goose-skin could be observed, neither on the chest nor on the neck. This experiment was repeated many times and always with the same result.

That the pilomotor nerves over a limited area may be rendered abnormally sensitive in cases of disease of an internal organ, in the same manner as the sensory nerves, appears likely from Case 5, and such observations as the following :—

Case 6.—Female, aged 59, has suffered from severe pains across the upper part of the abdomen occurring at intervals for the past twelve years. During the last three years she has suffered with increasing frequency, and for the last three months she has scarcely been without pain. She also suffers frequently from attacks of jaundice. I saw her in consultation, and as the symptoms pointed to gall-stone disease, recommended the advisability of an operation, for which purpose she was admitted into the Victoria Hospital, on October 31, 1893. Since her admission she has remained quite free from pain, and the jaundice from which she suffered is gradually disappearing. The attacks of pain are localized across the middle line of the abdomen, midway between the umbilicus and ensiform cartilage, extending well round to the right side. There is no cutaneous hyper-æsthesia; but there is an area of tenderness of indefinite extent, elicited by pinching the deeper structures of the abdominal walls (subcutaneous fat and muscles). On lightly rubbing the chest and abdomen with a piece of flannel, the goose-skin appears in a band three inches broad, extending across the abdomen above the umbilicus, to the left as far as the anterior axillary line, and to the right as far as the middle axillary. The area widens out on the right side, extending over the chest as high as the nipple, but not passing further back. A week after this observation, the patient having still been free from pain and the deep tenderness having nearly disappeared, the area of goose-skin obtained by stimulation was limited entirely to the right side, and covered an area less extensive than in the previous observation.

In another case of gall-stone disease under observation a somewhat similar relationship of exalted sensibility of the external body-wall and of the excitability of the pilomotor reflex can be demonstrated.

In several other cases, I have observed areas of goose-

skin limited to regions in which pain and hyper-æsthesia had been felt, but so far I have not yet been able to determine the exact harmony, or otherwise, of these manifestations. There is one feature about the distribution of these areas of goose-skin, that resembles the distribution of the hyper-æsthesia in visceral disease. Frequently the area does not correspond to the distribution of one or more spinal segments in their entirety, but spreads somewhat diffusely into portions of the area supplied by several spinal nerves. In my contribution on the sensory phenomena associated with visceral disease (BRAIN, Autumn Number, 1893, p. 321-374), I pointed out this characteristic both in the hyper-æsthesia of visceral disease and in that associated with herpes zoster. I am not sure whether the irritation tends to spread more along longitudinal paths, affecting the roots supplying the anterior, lateral or posterior aspects of the body (fig. 1), rather than to affect one spinal segment; but I incline to such a view. It may also be that whilst the segmentation of the cord is not so definite as to permit precise division of the cutaneous surface according to nerve supply, excitations spread from any given centre in the cord more readily along paths in a longitudinal than in a transverse direction.

A difference in the distribution of sensory and pilomotor nerves is also evident from the following observation. The chest of a woman whose breast had been excised a year before, presented a long cicatrix about the level of the third rib, extending from the sternum out into the axilla. On exposure of the skin to the air, the goose-skin spread over the chest and arm; but while it extended down to the upper margin of the cicatrix, it left quite free a band of about one-and-a-half inches below and parallel with the cicatrix. On testing the sensation of the skin below I found it quite normal. In another case, an adenoid tumour was removed from the breast. After stitching up the wound (the incision being about five inches in length and situated horizontally near the anterior axillary line), I observed the goose-skin spread over the whole chest, leaving an area about an inch broad internal to the line of incision quite unaffected; the skin was after-

wards found quite normal. The sensation caused by, or rather associated with the production of goose-skin, appears to be peculiar in its nature. Most people are acquainted with the sensation; and in Case 4 I made a curious observation. Once while testing for the goose-skin on the chest, I was looking for it near the point of stimulation. The patient called my attention to the fact of its production on the inner side of the upper arm. I asked him how he knew, and he said he felt it coming. On going further into this point, I found that, on certain areas of the chest being stimulated, he felt the sensation extend down the arm to the little finger where no goose-skin was visible. I asked him what kind of sensation it was, and he said, "It is a curious sensation as if goose-lumps were coming out." But when I called his attention to the fact that there was no such appearance on his wrist and hand, he examined the part and appeared surprised. Being somewhat doubtful of his veracity, I put him to the following test. I lightly stimulated the skin by drawing the pencil case transversely across the skin of the neck, gradually descending over the clavicle on to the chest; he said that no sensation whatever was produced; but directly I touched the skin below the first rib, he at once exclaimed that he experienced the sensation. It was also noticed that the sensation arose exactly where the line was crossed, which I had marked along the upper limit of the goose-skin on the chest. Similiarly, on testing the skin from the level of the umbilicus and gradually ascending whenever I touched the skin above the nipple, the same sensation was experienced. Excitation of any area between the nipple and first rib produced the feeling. The latter always quickly spread onwards from the chest to the inner surface of the arm, and down the ulnar border of the forearm to the little finger, occasionally with the accompaniment of goose-skin on the inner surface of the arm. The patient could have no knowledge of the distribution of the spinal nerves; but his sensation indicated their course so distinctly that I have no hesitation in giving credence to his expressions. The experiments were often repeated on both sides of the body, and these results verified.

My object in writing this paper is merely to call attention to a method of studying the nerve distribution, which is free from many objections that vitiate other methods. I would have liked to have compared the results obtained by clinical investigations, with those of Langley and Sherrington; but my observations are still far too few. From what I have seen, I feel convinced that very instructive results are obtainable, particularly with reference to the nerve distribution in the limbs, and to the diffusion of irritation to nerve centres in visceral disease. I must, however, remain content with presenting these suggestions, other matters preventing me from pursuing this inquiry for the present.

ONE HUNDRED CONSECUTIVE CASES OF EPILEPSY; THEIR REFRACTION AND THEIR TREATMENT BY GLASSES.[1]

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For some years past there has been a question constantly put forward as to a relationship between epilepsy and errors of refraction, which up to the present time has not been satisfactorily met.

With a view of collecting some certain information on the subject, I, last year, began examining the eyes of all the cases of epilepsy that I could obtain at the West End Hospital for Nervous Diseases and Paralysis, and treating them as far as possible with correcting lenses. I have the honour of submitting to you the results of this work in one hundred consecutive cases of epilepsy.

In order to remove all possible doubt as to diagnosis, each case has first been seen by the physician in attendance, and subsequently, at my request, passed on to me as being one of true epilepsy.

With regard to the examination of the eyes, I may state that in seventy-four cases sulphate of atropine drops (grs. iv. and grs. ii. ss. ad. ℥i.) were employed; in twenty-three cases hydrobromate of homatropine drops (grs. vi. ad. ℥j.) were used, and in three cases only the process was carried

[1] Read before the Ophthalmological Society and published in this Journal by permission of the Council.

through without a mydriatic. Of these three cases, one was a woman of sixty-seven years of age, with Hm. 1 D., and taking + 4.50 D. for near work; the second, a man with myopia, R.E.—8 D., L.E.—2 D., and the third was a patient, who strenuously objected to the drops, showing Hm. .50 D.

In working out the refractions, exceeding care has been taken to ensure accuracy in the results.

The first table contains the classification of the refractions of the one hundred cases. In this—

(*a*) The total refraction under the mydriatic has been taken.

(*b*) Hypermetropia of .75 D. has been reckoned as emmetropia.

(*c*) Astigmatism of .25 D. has not been included.

In the one hundred cases I have found the results shown in Table A.

Here the simple hypermetropia and the total astigmatism are equal in amount, each having forty-two cases.

Wishing to compare these results with the refractions obtaining in apparently normal eyes, I could find nothing more suitable for that purpose than my own paper, published in the last volume of the *Transactions of the Ophthalmological Society of Great Britain*. From the cases contained in this I have worked out the following percentages on the same lines, as I have done in the epileptic cases (see Table B).

The comparison between the two classes is shown there.

The marked differences exist in the amounts of the simple hypermetropia, and of the astigmatism. Of simple hypermetropia there are twenty-eight cases per cent. *less* in the epileptic than in the apparently normal class.

Of astigmatism of all kinds, there are twenty-six cases per cent. *more* in the epileptic division than in the normal one, this difference being in great part made up by the increase in epileptics of compound hypermetropic astigmatism, viz., eighteen cases more than the normal percentage. If the difference of twenty-six cases per cent. of total astigmatism in the epileptic be added to the forty-two cases per cent. of the simple hypermetropia of the normal class, the

result comes to within two cases per cent. of the normal amount of simple hypermetropia, viz., 68. It would almost appear as if the increase of astigmatism in epileptics were taken from the normal amount of hypermetropia.

I beg now to refer to the large table (C); on it will be found each case arranged transversely under its various headings, viz.:

(1) No. of case; (2) age; (3) sex; (4) family history as to fits; (5) age when fits began; (6) frequency &c., of fits; (7) duration of fits; (8) time when fits occur; (9) aura; (10) cephalalgia; (11) vertigo; (12) pupils; (13) fundus examination, &c.; (14) vision and refraction before mydriasis; (15) mydriatic used; (16) vision and refraction after mydriasis; (17) glasses ordered, &c.; (18) length of time glasses worn; (19) condition, &c., while wearing glasses.

Of one hundred consecutive cases of epileptics examined, twenty-five did not need glasses; seventy-five were ordered to wear them, and of these there were twenty-three who either did not wear them or failed to report themselves later, and could not be found. Of the remaining fifty-two patients there are—

(*a*) Thirteen who have had no fits since wearing the glasses, during periods varying from one year down to four months. These are Nos. 5, 11, 24, 25, 33, 52, 57, 58, 66, 78, 87, 90, 91.

(*b*) Three who have remained *in statu quo*, Nos. 3, 46, 72.

(*c*) Thirty-six whose condition has improved since using the glasses—in the majority of these the improvement is in a marked degree. The refractions of the thirteen cases who have had no fits, are shown in the large tables. With the exception of the one case of mixed astigmatism the errors are not great; the proportion of simple hypermetropia to the total astigmatism still stands about as in the larger scale, the compound hypermetropic astigmatism representing the greatest part as before.

It is not necessary for me to weary you with a long account of these cases. I will however read a few of them as examples; others may easily be found in the tables.

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Brief histories of some patients who have been free from fits since wearing glasses.

Case I., *No.* 5, *Sept.*, 1892.—A man aged 24 years, a porter by occupation. There is no family history as to fits. His fits began at the age of 21 years, and they have continued up to this month. He has about seven or eight in a month, and very rarely goes a month without an attack. He has often had several fits in rapid sequence. They always occur during the day—never at night. He has no aura. He complains of bad frontal headache and also of occipital pains; the latter are rare. Vertigo is not marked, but he has it occasionally. He has been taking bromide of potassium for a long while.

Oct., 1893.—He has had no fits since wearing his glasses, which were at first ordered for near work only. In July, his headaches still continuing, he was told to carry his spectacles always, and he is now absolutely free from cephalic pain. His general health is much better.

Case II., *No.* 11, *Sept.*, 1892.—A woman aged 20 years, a housemaid. Her mother has had fits since she was 9 years old up to the present date, Sept., 1892. A brother, aged 22 years, has had fits since 6 months. Her fits began at 19 years of age. She has now about one a month, but has had them more frequently. They generally occur at night. She has no aura, though on one occasion she saw "mixed colours" just before a fit. She suffers from vertical headache "dreadfully" and frequently; she also has attacks of vertigo nearly every afternoon. She has been taking bromide of potassium for a long time.

July, 1893.—She has sensations but no fits.

Sept., 1893.—She has slight sensations but no fits.

Case III., *No.* 24, *Nov.*, 1892.—A woman aged 30 years, a servant. There is no family history as to fits. Her fits began at 20 years of age. She has them at variable periods and very often. They occur usually in the morning, but also occasionally at night. She has no aura. She suffers frequently from headache, which is frontal and vertical in situation, also from numerous attacks of vertigo. She has been taking bromide of potassium for a long time.

June, 1893.—She has had no fits and has lost vertigo. The headache is rare and lessened in severity.

Sept., 1893.—No fits—no headaches to speak of—rarely some vertigo.

Case IV., *No.* 25, *Nov.*, 1892.—A youth aged 16 years, a tailor. There is no family history of fits. The fits began in childhood. He now has them about three times a week, usually at night. He has no aura. He suffers from frequent headaches which he calls "awful"; they begin with severe frontal pains, and then extends all over his head. Vertigo is common and usually occurs in the daytime. He has been taking bromide of potassium for a long time.

May, 1893.—No fits since glasses.

Sept., 1893.—No fits, no headaches and no vertigo.

Oct., 1893.—The same.

Brief histories of some cases of marked improvement.

Case I., *No.* 2, *Sept.*, 1892.—A woman aged 46 years, a dressmaker. Her eldest brother had fits between the ages of 14 and 21 years. Her fits began at about 18 years of age. She has one fit at variable periods; now they are much more frequent. She has occasionally gone a few months without an attack. She has them always at night. Her warnings are a "feeling of unsteadiness" in frontal and occipital regions of head. Her headaches are very severe and frequent, chiefly frontal, but also occasionally occipital, and her vertigo is so intense that she reels in her walk sometimes. She has taken bromide of potassium for a long time.

March, 1893.—Has had two fits since glasses; ordered an additional pair for presbyopia.

June, 1893.—No fits, no cephalalgia of importance.

July, 1893.—No fits, slight cephalalgia.

Sept., 1893.—One very slight fit in August. Is much better in general health.

Case II., *No.* 28, *Nov.*, 1892.—A girl aged 15 years. There is no family history as to fits. The fits began at 14 years of age. She has now about one attack a week—often two in rapid sequence. They occur at night towards the early morning. She has no aura. Vertical headache is very frequent and she occasionally has occipital pains. She is never giddy. She has been taking bromide of potassium for a long time. She went from Nov., 1892, quite without fits to Sept. 13, 1893, when she had one attack, being at this time extremely worried and nervous about obtaining a situation.

Case III., *No.* 16, *October*, 1892.—A girl aged 14 years. Her father is believed to have had fits in early youth. Her fits began at 9 years of age, when she had one fit. She was then free from

attacks until the beginning of her fourteenth year, when they appeared again. She is now having six or seven fits during the day, and they occur about every hour during the night. She has no aura, and she suffers from very severe and frequent headaches, which are frontal and vertical in position; she has no marked vertigo; she has been taking bromide of potassium for some time. Her mother objected to her reading because she thought the fits were brought on through it.

August, 1893.—No fits since glasses; no cephalalgia; is much better.

September 4, 1893.—Has had two fits during the night; she is near her menstrual period.

September 15.—She has had a few fits at night.

Case IV., *No.* 20, *Oct.*, 1892.—A man aged 20 years, a carpenter. His mother has about one fit a month. His fits began at 16 years of age. He has now about one a week; they occur in the daytime, never at night. He has no aura; he is free from cephalalgia, except just after a fit when it is frontal, but this is nothing marked. There is never any vertigo. He has been taking bromide of potassium for a long time.

May, 1893.—No fits since glasses.

Sept., 1893.—One very slight fit.

In all the cases the physician has, I believe, continued his usual treatment for some time after the glasses had been ordered.

Case II. of this second series is a type of several that I have met with. The proper correction of the error of refraction by glasses evidently arrests their fits, and they do well until some other irritation comes to play. In this case the patient has no fits from the time she begins to carry glasses, from November, 1892, to September, 1893, she then has one fit as a result of nervous excitement produced by a difficulty in obtaining a certain situation. Another patient also has practically the same history, and brings on his fit by foolishly diving from a height into cold water. Another one is free for months and then is nearly run over by a hansom cab, when she immediately has six fits. Another goes many months and has a return of her fits just at her menstrual period. In a great number some such occasion appears to cause a return of the fits, but in no such case have the fits resumed their previous severe character or frequency.

It seems to result that, given a certain condition of instability of the nervous system—

(*a*) Errors of refraction may excite epilepsy.

(*b*) The correction of the errors of refraction will—in combination with other treatment—in many cases cure or relieve the epileptic condition. In this hundred cases forty-nine patients have been cured or relieved.

(*c*) In some cases, when the refraction error has been corrected the epilepsy will continue, generally in a modified form, in consequence of other irritation, even though the error of refraction may have been the exciting cause of the fits in the first instance, as shown in the cases that I have pointed out.

I am strongly of the opinion that in every case of epilepsy—in addition to general treatment and the investigation of other organs—the eyes should be carefully examined under a mydriatic, with a view to correcting any error of refraction that may exist by proper spectacles.

A few other points occur which I wish to notice, viz. :—

1. That in twenty-three cases only could I obtain any family history as to fits.

2. The marked severity and frequency of frontal headache, I found about sixty-five per cent. suffered from this ; vertical headache came next in number but never appeared so severe as the frontal form.

3. That in about sixty-seven cases the pupils were more or less dilated, and in a large proportion of these, their reaction was lazy; in about six cases the pupils were more or less contracted, and the remaining twenty-seven patients appeared to be normal in every way.

4. That in five cases I found lens opacities of apparently congenital origin.

5. That pulsating veins and physiological cups were very frequently observed.

TABLE A. TABLE B.

Epileptics, per cent. from 100 cases.	TABLE OF COMPARISON BETWEEN EPILEPTICS AND APPARENTLY NORMAL CASES.	Normals, per cent. from 50 cases.
7	Emmetropia	6
42	Simple Hypermetropia	70
6	Simple Myopia	2
	Total Astigmatism: 3 Simple Hyper., Astigmatism 4	
	Total Astigmatism: 24 Compound do. do. 6	
42	Total Astigmatism: 2 Compound Myopic do. 0	16
	Total Astigmatism: 6 Mixed Astigmatism 0	
	Total Astigmatism: 7 Other Astigmatism (marked Anisometropia) 6	
3	Other Cases (marked Anisometropia)	6
100	TOTAL	100

CHARTS, PAGES 542-555, BEST COPY AVAILABLE.

No of Case.	Sex.	Age.	Family History as to Fits.	Age when fits began	Frequency, &c., of Fits before Examination.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fundus,	[illegible] and before
1	M	8	Aunt died at 35 years of age from epileptic fits	7	Has now about 5 or 6 fits a week. Has had 5 fits in one day	4 or 5 min.	Matutinal, between 6 & 8 o'clock	Occasionally feels giddy before a fit	Frontal, frequent and severe	Occasionally before a fit	Moderately dilated	Pulsati veins	R. ∫ L. ∫
2	F	46	Eldest brother had fits from 14 to 21 years of age	18	Has fits at variable periods. Occasionally has gone a few months. Now they are more frequent	A few min.	Nocturnal, never in daytime	Feelings of unsteadiness in frontal region	Frontal and occipital, very severe & frequent	Frequent: reels often in her walk	Moderately dilated	Discs hy anæmic, phy logical c pulsati veins, pe pheral st in both le (congenita	A – 2 n.i.
3	M	25	One sister had fits in youth, beginning at 12 years of age	10	Has about 50 fits per annum. Occasionally 3 or 4 in rapid sequence	About 3 min.	Nocturnal	Fit begins by head turning in small jerks to left shoulder	Frontal, not severe and occasional	None	Normal	Pulsati veins, pe pheral str both le (congenita	R. ½ L. ½
4	M	16	.....	12	.....	.....	.....	.....	.....	.....	.....	.....	R. ∫ le L. ∫ de
5	M	24	None	21	Has 7 or 8 fits in a month. Rarely goes a month without one. Occasionally 3 or 4 in rapid sequence	2 or 3 min.	Daytime, never nocturnal	None	Frontal, severe and frequent	Occasionally	Dilated, slow reaction	Pulsati veins. R physiolog cupping. L —nil.	∫ H.m. ∫ H.m.
6	M	15	None	7	Has 2 fits in a week	5 min.	Matutinal, never nocturnal	Peculiar feeling in eyes	Frontal, not bad; occasional	Rarely	Moderately dilated	Normal	∫ H.m. ∫ de
7	F	45	.....	43	.....	.....	.....	.....	.....	.....	.....	.....	∫ H.m. ∫ H.m.
8	F	17	None	11	Has about 4 fits in a month. Occasionally 2 or 3 in rapid sequence	2 or 3 min.	Nocturnal	Pains in legs, and occasionally sees patches of green going into red	Frontal, frequent and severe, especially bad before a fit	Frequent	Dilated	Discs hy anæmic, phy logical c Y-sha opacities aut., cap lenses	R. ∫ n. L. ½ n
9	F	7	None	3	Has 3 or 4 fits daily for some days, then perhaps goes a month. Cannot walk. Sits where placed and never moves	A few min.	Nocturnal & day	None	Frontal, frequent and severe		Moderately dilated	Normal	Cannot r
10	F	35	None	17	Daily	A few min.	Nocturnal, rarely in daytime	Sensations in right arm	Frontal and occipital rare, & only after a fit not severe	Very rarely	Normal	Normal	R. ∫ n.i. L. ∫ iv. r
11	F	20	Mother has had fits since 9th year, one brother since 22nd year (6 mos.)	19	Has fits about once or twice a month, has had them more frequently	10 min.	Nocturnal	None (once had "mixed colors" before eyes)	Vertical, frequent and "dreadfully"	Often, nearly every afternoon	Moderately contracted	Discs hy mic, s pulsat veins	Hm.] D. Hm.] D.
12	F	17	None	15	Has about once in 3 months an attack of 20 or 30 fits in one week	10 min.	Nocturnal & daytime	None	Frontal	Matutinal on rising	Moderately dilated	Pulsating physiol. c	R. ∫ n.i. L. ∫ n.i.

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Vision and Refraction before Mydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
R. $\frac{6}{9}$ n.i. L. $\frac{6}{9}$ let n.i.	Gutt. Atrop. Sulph. grs. liss. ad ℥ i. thrice daily for 3 days	$<\frac{6}{60}$ + 3.50 D. = $\frac{6}{9}$ $<\frac{6}{60}$ + 4.50 D. = $\frac{6}{9}$	+ 2.50 D. + 3.50 D. } for constant wear	3 mos.	JULY—Fits are many less in number. Cephal. and vertigo better. SEPT.—Still better. Mother has not kept an account, but he often goes many days without a fit (glasses not obtained until months after being ordered)
$\frac{6}{18}$ − 2.75D. = $\frac{6}{12}$ L. $\frac{6}{9}$ n.i.	Do. grs. iv. ad ℥ i	$\frac{6}{18}$ + + 1.50 D. emm. = $\frac{-.75 \text{ D.} = \frac{6}{9}}{+1 \text{ D. cyl. ax. } -}$ $\frac{6}{18}$ + + 1.75 D. emm. = $\frac{0 = \frac{6}{9}}{+1 \text{ D. cyl. ax. } \diagdown 120°}$	(1) Less + 1 D. sph. for distance. (2) c + 2 D. sph. for near work	12 mos.	During the first 6 months she had glasses for distance only, and had 2 fits then. Subsequently she had another pair of spectacles for near work for presbyopia, since when she has had one slight sensation rather than a fit, and her bad headaches are almost gone. Vertigo is also rare. Is much better and stouter
R. $\frac{6}{9}$ n.i. L. $\frac{6}{24}$ n.i.	Do. 7 days	$<\frac{6}{60}$ + + 1 D. + 2.50 D. = $\frac{+1.75 \text{ D.} = \frac{6}{9}}{+1.75 \text{ D. cyl. ax. } \mid \text{v.}}$ $<\frac{6}{60}$ + + .75 D. + 3 D. = $\frac{+1.50 \text{ D.} = \frac{6}{9}}{+2.50 \text{ D. cyl. ax. } \mid \text{v.}}$	Ordered less + 1 D. sph. for constant wear	8 mos.	In MARCH fits not so severe in character. During 5 months before glasses 21 fits, during 5 months after 12 fits. In MAY case died—cause unknown
R. $\frac{6}{9}$ let n.i. L. $\frac{6}{9}$ do.	Do. 3 days	$\frac{6}{12}$ let + + 1.75 D. + 2 D. $\frac{6}{24}$ + + 2 D. + 1.75 D.	No glasses.		
R.m. 1 D. = $\frac{6}{9}$ R.m. 1 D. = $\frac{6}{9}$	Do	+ 2.75 D. = $\frac{6}{9}$ + 2.75 D. = $\frac{6}{9}$	+ 1.75 D. sph. for near work, then constant	12 mos.	Has had no fits since wearing glasses. Glasses were first ordered for near work only, but because of cephal. he was told to carry them constantly, since when cephal. has entirely disappeared
R.m. 1 D. = $\frac{6}{9}$ do.	Do.	$\frac{6}{12}$ + 1.75 D. = $\frac{6}{9}$ $\frac{6}{24}$ + 1.75 D. = $\frac{6}{9}$	+ 1 D. constant wear	11 mos.	JUNE—Glasses confirmed. JULY—One slight fit. Has worn glasses for near work only
R.m. .50 D. = $\frac{6}{9}$ L.m. 1 D. = $\frac{6}{9}$	Homatropine	+ 1 D. = $\frac{6}{9}$ let + 1.75 D. = $\frac{6}{9}$	+ 2 D. + 2.50 D. } For near work		
R. $\frac{6}{9}$ n.i. L. $\frac{6}{18}$ n.i.	G. A. S. grs. iv. 3 days	+ + 2 D. + 1.25 D. = $\frac{+1.25 \text{ D.} = \frac{6}{9}}{+.50 \text{ D. cyl. ax. } \diagup 110°}$ + + 1.25 D. + 2 D. = $\frac{+1.25 \text{ D.} = \frac{6}{9}}{+1 \text{ D. cyl. ax. } \diagdown 150°}$	Cylinders only for constant wear	6 mos.	JUNE—Has had 4 fits only since wearing glasses, these all upon one day. SEPT.—Had 1 fit JULY 1st and 1 AUG. 19th. Does not wear spectacles constantly
Cannot read.	Do. grs. liss. ad ℥ i. 1 week	+ + 4 D. + 2.50 D. = $\frac{+2.25 \text{ D.}}{+1.50 \text{ D. cyl. ax. } \mid \text{v.}}$ + + 4 D. + 2.25 D. = $\frac{+2 \text{ D.}}{+1.50 \text{ D. cyl. ax. } \mid \text{v.}}$	Ordered less + 1 D. sph. for constant wear	12 mos.	JUNE—Decidedly better, walks fairly well, is fatter and better tempered. Fits less in number and all of lessened severity. SEPT. — Has been 4 months without a fit. Has recently had 3 fits. Is now more intelligent and altogether better in health
R. $\frac{6}{9}$ n.i. L. $\frac{6}{9}$ iv. n.i.	Homatropine	+ 1 D. = $\frac{6}{9}$ + 1 D. = $\frac{6}{9}$ iii.	No glasses		
R.m. 1 D. = $\frac{6}{9}$ let. L.m. 1 D. = $\frac{6}{9}$ let.	G. A. S. grs. iv. ad ℥ i. t. d. ut. 3 days	$\frac{6}{18}$ = + + 1.50 D. + 1.50 D. + 2.50 D. + 1 D. cyl. ax. R. ⧹ 45° = $\frac{6}{9}$ iv. L. ⧸ 55° $\frac{6}{60}$ = Do.	Ordered less + 1 D. sph. for constant wear	12 mos.	JULY—Still has sensations, but no fits. SEPTEMBER—no fits, still sensations
R. $\frac{6}{9}$ n.i. L. $\frac{6}{9}$ n.i.	Do.	+ .75 D. + 1 D. Do.	No glasses		

XUM

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when fits began	Frequency, &c., of Fits before Examination.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fundus.	z and ore M
13	F	16	None	6	Has one or more fits daily	15 min.	Nocturnal, rarely in daytime	None	Frontal, very severe and frequent	None	Normal	—	R. $\frac{5}{5}$ L. $\frac{5}{5}$
14	M	27	.....	19	One fit in 3 days, has gone 14 days without one	—	Nocturnal	None	—			—	R. $\frac{5}{5}$ le L. $\frac{5}{5}$ le
15	F	16	.....	15	.....	.....	.....	.....	.....	.....	.....	.....	R. $\frac{5}{5}$ L. $\frac{5}{5}$
16	F	14	? Father when a child	9	One fit at 9 years of age, no more till 14. Began again and still continue. Hourly during night and 6 or 7 in the daytime	A few min.	Nocturnal & in daytime	None	Frontal and vertical, very severe and frequent, also very bad after a fit	None	Normal	Pulsating v	R. $\frac{5}{5}$ L. $\frac{5}{5}$
17	F	39	None	25	Has about 1 fit every 3 weeks, formerly more frequent	A few min.	Formerly nocturnal, now nearly always in the daytime	None	Not often frontal, shooting neuralgic pains	None	Moderately dilated	Pulsating v R. E. — whitestreg cup. L —do hyp mic	R. $\frac{5}{5}$ L. $\frac{5}{5}$
18	F	18	Mother had fits when a child	13	Has 3 or 4 fits a week	5 min.	Nocturnal, rarely in daytime	None	None, a "dulness" after a fit	None	Dilated, slow reaction	Pulsating v marked regularity edges of	H.m. .5 D
19	F	36	None	16	Has about 15 fits a year. Formerly has had 8 fits a day	10 min.	Nocturnal, rarely in daytime	Frontal cephalalgia, has seen lights before a fit	Frontal very bad before a fit	Formerly just before fits, now less	Moderately dilated	Discs hyp mic, sl physiol. pings, p ting vein	H.m.
20	M	20	Mother has about 1 fit a month	16	Has about 1 fit a week	10 min.	Matutinal	None	None, except after a fit, then frontal but not bad	None	Dilated	Physiol. pings, p ting veins	R. $\frac{5}{5}$ n L. $\frac{5}{5}$ n
21	M	41	One brother went insane and died from fits	32	Has had about 16 fits in all	A few min.	Matutinal	None	Vertical, but not marked	Very rarely	Normal	Pulsating v	H.m. 1 D
22	M	26	None	13	Has 5 or 6 slight fits daily & several bad ones during a week	2 or 3 min.	Matutinal, also nocturnal	None	Frontal, severe, also occipital	Constantly, & then his sight "appears to go"	Moderately dilated		H.m. 1 D
23	F	35	None	30	Has 2 or 3 fits a day; has had 7 in 1 hour one day. Rarely goes a month without an attack	Under $\frac{1}{2}$ hour	Matutinal	Feeling in right hand and leg occasionally	Frontal, always bad	Constantly, nearly every morning	Moderately dilated, slow reaction	Discs whit pulsat veins	R. $<\frac{5}{5}$ L. $<\frac{5}{5}$
24	F	30	None	20	Has fits at variable periods, but frequently	A few min.	Matutinal, also nocturnal	None	Frontal and vertical frequent	Frequent	Moderately dilated	Pulsat veins	$\frac{5}{5}$ ii. $\frac{5}{5}$ iii.
25	M	16	None	In babyhood	Has fits about 3 times a week	A few min.	Nocturnal	None	Frontal, extending all over; at all times "awful"	Very frequent	Dilated, slow reaction	Pulsat veins	H.m. 1 D Do.
26	M	21	None	15	Has from 1 fit a week to 1 in two months	A few min.	Matutinal	None	None	None, unless when excited	Moderately contracted	Pulsati veins. L. Central pl cup.	H.m. 1.50 Do.
27	F	44	None	29	Has 3 or 4 fits a day, with occasional intervals of a week or so	3 or 4 min.	Daytime, occasionally nocturnal	Sensations in legs occasionally	Pressure on vertex	Frequent & severe	Normal	—	H.m. .50 H.m. .50

XUM

Vision and Refraction before Mydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
R. $\frac{6}{24}$ n.i. L. $\frac{6}{12}$ n.i.	Do. grs. fiss. ad ℥ i. do.	$\frac{6}{24}$ + −.75 D. + 3.50 D. = + 3.50 D. / − 4 D. cyl. ax. − = $\frac{6}{12}$ $\frac{6}{24}$ + + 4 D. − .75 D. = + 4 D. / − 4 D. cyl. ax. − = $\frac{6}{12}$	Ordered less + 1 D. sph. for constant wear	10 mos.	JULY—No fits until DECEMBER, when she had rheumatic fever, therefore discontinued spectacles for more than 14 days. The fits returned almost daily for some weeks and then slowly disappeared. She has now been some weeks free. Cephal. is rare now
R. ∫ let n.i. L. ∫ let n.i.	Homatropine	+ 1.25 D. = ∫ let Do.	No glasses		
R. $\frac{6}{24}$ n.i. L. $\frac{6}{18}$ n.i.	G. A. S. grs. iv. ad ℥ i. t. d. ut. 3 days	+ 2.75 D. = ∫ let / + 4 D. cyl. ax. − + 2.75 D. = ∫ / + .50 D. cyl. ax. −	Ordered less + 1 D. sph. for constant wear		
R. $\frac{6}{24}$ n.i. L. $\frac{6}{18}$ n.i.	Do. 7 days	$\frac{6}{24}$ + − ? + 4.50 D. = + 4.50 D. / − 5 D. cyl. ax. − = $\frac{6}{12}$ $\frac{6}{18}$ + − 5 D. − 1 D. = −.50 D. / − 4 D. cyl. ax. − = $\frac{6}{12}$	+ 3.50 sph. − 5 D. cyl. ax. − − 1.50 D. − 4 D. cyl. ax. − For constant wear	12 mos.	MARCH—No fits since glasses no cephal., feels well, looks bright and lively. OCTOBER—Had 1 fit on 4th September, 1893, had 2 during the night, having previously had no fits since glasses, that is, during 11 months. She began fits again at a menstrual period, and they still occur occasionally but are slight in character
R. $\frac{6}{12}$ n.i. L. $\frac{6}{12}$ n.i.	Homatropine	$\frac{6}{6}$ × + 3.50 D. emm. = 0 / + 3.50 D. ax. \ 45° = $\frac{6}{6}$ $\frac{6}{12}$ × + 3.50 D. − 1.50 D. = 0 / + 3.50 D. ax. / = $\frac{6}{12}$ let	No glasses (afraid to wear them)		
H.m. .50 D. = ∫ Do.	G. A. S. grs. iv. ad ℥ i. t. d. ut. 3 days	+ + 1.50 D. + .75 D. + + 1.25 D. + 1.75 D.	No glasses		
H.m. .50 D. = ∫ Do.	Homatropine	+ 1.25 D. = ∫ + 1.25 D. = ∫ iv.	No glasses		
R. ∫ n.i. L. ∫ n.i.	G. A. S. grs. iv. ad. ℥i. t.d. ut. 3 days	+ + .75 D. + 2 D. = + .75 D. / + 1.25 D. ax. | v. = ∫ + + .75 D. + 2.25 D. = + .75 D. / + 1.25 D. ax. / 100° = ∫	Ordered cylinders only for constant wear	12 mos.	Had no fits from time of wearing glasses (for 11 months), when he had 1 slight fit. Feels much better
H.m. 1 D. = ∫ let Do.	Homatropine	$\frac{6}{12}$ + + 1.25 D. + 1.50 D. = ∫ let Do.	No glasses		
H.m. 1 D. = ∫ Do.	G. A. S. grs. iv. ad ℥ i. t. d. ut. 3 days	+ + 2.75 D. + 3.75 D. = + 2.75 D. / + 1 D. cyl. ax. | v. = ∫ let + + 2.75 D. + 3.50 D. = + 2.75 D. / + .75 D. cyl. ax. | v. = ∫	Ordered less + 1 D. sph. for constant wear		JUNE—Has had no fits since atropine drops were begun. Cephal. better
L. < $\frac{6}{6}$ n.i. < $\frac{6}{6}$ n.i.	Do.	< $\frac{6}{6}$ + + 1.75 D. = $\frac{6}{6}$ / − 4 D. cyl. ax. \ 50° < $\frac{6}{6}$ + 1 D. = $\frac{6}{12}$ let / − 3 D. cyl. ax. / 60°	Ordered cylinders only for constant wear		Glasses obtained 14th April, 1893. JULY—Has had 4 fits since glasses were worn, and cephal. is very much better. Vertigo is rare. SEPTEMBER—Three fits; very much better in health
L. $\frac{6}{12}$ ii. n.i. < $\frac{6}{6}$ iii. n.i.	Do.	< $\frac{6}{6}$ + + 2.25 D. + 4.50 D. = + 2.25 D. / + 2.25 D. cyl. ax. | v. = $\frac{6}{6}$ < $\frac{6}{6}$ + + 2.50 D. + 4.50 D. = + 2.25 D. / + 2 D. cyl. ax. | v. = $\frac{6}{6}$	Ordered less + 1 D. sph. for constant wear	11 mos.	Is better. Has had no fits since wearing glasses. Cephalalgia is very rare. Vertigo has almost gone
H.m. 1 D. = ∫ let Do.	Do.	$\frac{6}{24}$ + 3 D. = $\frac{6}{6}$ $\frac{6}{36}$ + 3 D. = $\frac{6}{6}$	+ 2 D. for constant wear	11 mos.	Is better. No fits since glasses, no cephalalgia and no vertigo
H.m. 1.50 D. = ∫ Do.	Do.	$\frac{6}{24}$ + 3.50 D. = ∫ let $\frac{6}{12}$ let Do. = ∫ let	+ 2 D. for constant wear		SEPTEMBER—Is much better, but has not worn glasses constantly until after JULY, since when he has had no fits
H.m. .50 D. = ∫ iii. H.m. .50 D. = ∫ ii.	None	—	+ 1 D. for near work		

XUM

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when fits began	Frequency, &c., of Fits before Examination.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fundus, &c.
28	F	15	None	14	Has 1 fit a week, often 2 or 3 in rapid sequence	10 min.	Nocturnal & early morning	None	Vertical frequent, occipital occasional	None	Dilated	Pulsati veins
29	F	34	None	22	Has about 1 fit in 14 days	A few min.	Nocturnal, also in daytime	Heavy and sleepy before fits	Frontal very frequent, also universal after a fit	Frequent	Normal	Pulsati veins. R. Phys. cup
30	F	17	None	13	Has about 1 fit a month, at variable periods	15 min.	Both in daytime and night-time	None	Frontal, bad after a fit	Not marked	Dilated, slow reaction	Pulsati veins, sl phys. cup
31	F	16	Maternal grandmother had fits which stopped at 16 years of age	13	Has from 2 fits a day to 2 a week. Has gone 2 mos. without a fit	10 min.	Nocturnal, also in daytime	None	Frontal frequent and severe, occasional temporal	None	Moderately dilated	—
32	M	16	None	12	Has 1 fit daily, occasionally has attacks of many fits and then goes a week or so without any	5 min.	Nocturnal, also in daytime	None	Vertical, occasionally not severe	Rarely	Moderately dilated	Pulsati veins, ce phys. cup
33	M	65	None	57	Has about 1 fit a week, rarely goes 2 weeks without one	A few min.	Nocturnal, rarely in daytime	Often sees colored lights before a fit	Frontal not very marked	Occasionally	Moderately contracted	Normal
34	M	17	None	6	Has 3 or 4 fits a day, has had 20 fits in one day	About 2 min.	Both at daytime and at night	None, except perhaps a little weakness in legs	Frontal marked after a fit	None	Moderately dilated	Phys. c pulsati veins. R. has small city (coc tal)
35	F	26	None	10	Has 3 or 4 fits a year, formerly they were more frequent	½ hour	Nocturnal	None, unless irritability	Vertical severe and frequent; occipital rarely	Frequent	Moderately dilated	Phys. c pulsati veins
36	F	32	None	30	Has 3 or 4 fits a month, formerly had 3 or 4 in a day	20 min.	Nocturnal, sometimes in the daytime	None	None, except occipital: rarely after a fit	None	Normal	Pulsati veins
37	M	16	None	15	Has 3 or 4 fits a week	A few min.	Day also nocturnal	A "giddiness in the head"	Frontal frequent	Matutinal frequent and bad	Normal	Pulsati veins
38	F	35	None	24	Has 1 fit in a month, formerly many fits and attacks of numbers of fits in sequence	15 min.	Nocturnal, rarely in the daytime	"Nervous feeling"	Frontal bad & frequent, also occipital	Constantly matutinal	Moderately dilated	Pulsati veins
39	F	17	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
40	M	17	None	2	Has 1 or 2 fits a day, rarely has gone a month or so without an attack	5 min.	Both at daytime and at night	Strange feelings inside	Frontal and temporal	Occasionally	Normal	Pulsati veins and tral st phys. cup
41	M	15	None	14	Has 2 fits a day	3 or 4 min.	Nocturnal	Rarely a feeling of unsteadiness	Frontal occasional but bad	2 or 3 times a day	Normal	Central cup., ting vein
42	M	15	None	12	Has 1 fit a month	3 or 4 min.	Daytime	None, (unless "irritable")	Frontal not frequent or severe	Rare	Normal	
43	F	28	None	12	Has 6 or 7 fits during the day, and some attacks of 5 or 6 in rapid sequence	2 or 3 min.	Both at daytime and at night	Sometimes a slight pain in left hand	Occipital severe and frequent, vertical after a fit	None	Dilated	Phys. c pulsati veins

Vision and Refraction before Mydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
R. $\frac{6}{18}$ n.i. L. $\frac{6}{24}$ .i.	G. A. S. grs. iiss. ad ℥ i. t. d. ut. 7 days	$<\frac{6}{60}$ + + 1.75 D. = + 1.75 D. = $\frac{6}{12}$ + 6 D. = + 4 D. cyl. ax. | v. $<\frac{6}{60}$ + + 1.25 D. = + 1.25 D. = $\frac{6}{12}$ + 6.50 D. = + 4 D. ax. | v.	Ordered cylinders only for constant wear	11 mos.	JUNE—No fits since glasses; much better. AUGUST—Ditto. SEPTEMBER—One fit, was much excited about obtaining a situation
R. $\frac{6}{9}$ n.i. L. $\frac{6}{9}$ n.i.	Do. grs. iv. ad ℥ i.	$\frac{6}{24}$ + 1.75 D. = $\frac{6}{9}$ $\frac{6}{24}$ + 1.25 D. = $\frac{6}{9}$	Refused glasses + 1 D. for near work		
R. $\frac{6}{9}$ let n.i. L. $\frac{6}{9}$ let n.i.	Do.	$\frac{6}{9}$ iv. + 1 D. = $\frac{6}{9}$ $\frac{6}{9}$ iv. + 1 D. = $\frac{6}{9}$	No glasses		
R. $\frac{6}{9}$ n.i. L. $\frac{6}{9}$ n.i.	Do.	$\frac{6}{12}$ + 1.50 D. = $\frac{6}{9}$ let. $\frac{6}{12}$ let. + 1.50 D. = $\frac{6}{9}$ let.	No glasses		
R. $\frac{6}{9}$ H.m. 1 D. = $\frac{6}{9}$ L. $\frac{6}{9}$ Do.	Do.	$\frac{6}{12}$ + 1.25 D. = $\frac{6}{9}$ $\frac{6}{12}$ let. + 1.25 D. = $\frac{6}{9}$	No glasses		
R. $\frac{6}{9}$ H.m. 1.25 D. = $\frac{6}{9}$ let L. $\frac{6}{18}$ Do.	Homatropine	+ 1.50 D. = $\frac{6}{9}$ let. Do.	+ 1 D. for distance, + 5.50 D. for near work	10 mos.	JUNE—No fits since glasses, though still sensations. Ceph. and vertigo are much better SEPTEMBER—No fits, still has occasional sensations. No ceph. and vertigo is rare now
R. $\frac{6}{9}$ n.i. L. $\frac{6}{18}$ n.i.	Gutt. atrop. sulph. grs. iv., ad ℥i. t.d. ut. 3 days	+ 2 D. = $\frac{6}{9}$ + 1.25 D. = $\frac{6}{9}$	+ 1 D. } for near + .50 D. } work	4 mos.	JULY—Is much better. SEPTEMBER—No fits since JULY till 18th inst., when one took place
R. $\frac{6}{18}$ let n.i. L. $\frac{6}{9}$ let n.i.	Do.	+ + 3.50 D. + 3.50 D. = $\frac{6}{9}$ + 5 D. + 1.50 D. cyl. ax. \ 60° + + 3 D. + 3 D. = $\frac{6}{9}$ + 4 D. + 1.50 D. cyl. ax. \ 160°	Ordered less + 1 D. sph. for constant wear	10 mos.	Health is much better. Ceph. much improved. Has had 3 fits during this time
R. $\frac{6}{9}$ let H.m. 1 D. = $\frac{6}{9}$ L. $\frac{6}{18}$ let n.i.	Do.	$\frac{6}{18}$ + 2.25 D. = $\frac{6}{9}$ $\frac{6}{18}$ + 2.25 D. = $\frac{6}{9}$	+ 1 D. for constant wear	10 mos.	SEPTEMBER — Has not worn glasses regularly. Fits many less in number. Ceph. better
R. $\frac{6}{9}$ L. $\frac{6}{9}$	Do.	$\frac{6}{18}$ + 2 D. = $\frac{6}{9}$ $\frac{6}{18}$ + 2 D. = $\frac{6}{9}$ = $\frac{6}{9}$ let.	+ 1 D.	6 mos.	JUNE—Now has 1 fit a month. Ceph. better. Ordered to wear spectacles constantly
R. $\frac{6}{9}$ let L. $\frac{6}{9}$ iii.	Do.	+ .50 D. = $\frac{6}{9}$ + 2 D. cyl. ax. | v. + 1 D. = $\frac{6}{9}$ + 1.50 D. cyl. ax. / 55°	Ordered less + 1 D. sph. for constant wear	—	Has not worn glasses and remains *in statu quo*
R. $\frac{6}{18}$ n.i. L. $\frac{6}{18}$ n.i.	Do.	$\frac{6}{12}$ i. × + .50 D. 0 = $\frac{6}{9}$ let. + 1.75 D. + 2.25 D. cyl. ax. | v. $\frac{6}{12}$ i. × + 2.25 D. 0 = $\frac{6}{9}$ i. .75 D. + 3 D. cyl. ax. | v.	Ordered $\bar{c}$ — .50 D. sph. for constant wear		
R. $\frac{6}{9}$ let L. $\frac{6}{9}$ n.i.	Do.	$\frac{6}{9}$ + .50 D. = $\frac{6}{9}$ let. $\frac{6}{12}$ i. + .75 D. = $\frac{6}{9}$ let.	No glasses.		
R. $\frac{6}{9}$ H.m. .50D. = $\frac{6}{9}$ let L. $\frac{6}{24}$ Do. $\frac{6}{9}$	Do.	$\frac{6}{9}$ + 1.50 D. = $\frac{6}{9}$ $\frac{6}{24}$ + 1.50 D. = $\frac{6}{9}$	No glasses.		
R. $\frac{6}{9}$ H.m. .50D. = $\frac{6}{9}$ let L. $\frac{6}{24}$ Do. $\frac{6}{9}$	Do.	$\frac{6}{18}$ + 2 D. = $\frac{6}{9}$ $\frac{6}{24}$ + 2 D. = $\frac{6}{9}$ = $\frac{6}{9}$ let.	+ 1 D. for near work	9 mos.	JULY—Has not worn glasses regularly; says he is much better. SEPTEMBER—Has worn glasses. Has had no fits
R. $\frac{6}{9}$ iii. H.m. 1 D. = $\frac{6}{9}$ L. Do.	Do.	$\frac{6}{60}$ + 3.50 D. = $\frac{6}{9}$ iii. $<\frac{6}{60}$ + 3.50 D. = $\frac{6}{9}$	+ 2 D. for constant wear	9 mos.	MARCH—Has had 1 mild fit since glasses. APRIL—No fits. MAY—Six slight fits in sequence (frightened by cab). JULY—No fits. Ceph. rare now. SEPTEMBER—Has had 4 slight fits in one morning. Mother says she is better in every way

XUM

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when Fits began	Frequency, &c., of Fits.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fundus, &c.	...ion an...before
44	M	25	None	11	Has 1 fit a week	A few min.	Matutinal, has had 1 onlyatnight	None	Vertical and parietal, severeand frequent	Frequent	Moderately dilated, slow reaction	Central phys. cup., pulsating veins. L. ant. cup; lens has a congenital plaq...	R. ...– 3 L. ...– 2
45	M	18	None	15	Has 1 fit a month	20 min.	Daytime	Hissing in the ears about 5 minutes before a fit	None	Very rare	Normal	Pulsating veins	R. ...let. = L. ...let. =
46	M	16	None	13	Has 2 or 3 fits every night	3 or 4 min.	Nocturnal	"Funny feelings in stomach, forehead and eyes"	None	Rare, sometimes before and after a fit	Moderately dilated	Pulsating veins. R. phys. cup.	R. ... L. ...
47	M	31	None	24	Has 1 fit at variable periods, but many sensations, formerly many more, about 1 or more in 14 days	A few min.	Daytime	Singing in ears and giddiness	None	None, exceptjust before a fit	Dilated	—	R. ...– 3. L. ...– 3
48	M	35	None	11 mos.	Daily, several, has gone 3 weekswithout	3 min.	Nocturnal, seldom in day	Pain in right side of body and singing in the ears	Temporo-frontal constant	Rare	Moderately dilated, slow reaction	Discs hyper... slight pulsating veins. L.E. small phys. cup.	R. ... L. ...
49	F	14	None	3	Has 1 fit a month, from 3 to 13 years of age, no fits	A few min.	Matutinal	None	Frontal frequent, but only severe after a fit	Occasionally very bad	Moderately dilated	Discs hyper... pulsating veins. R.E. small phys. cup.	R. ... L. ...
50	M	11	None	9	Has 2 fits a week, has gone 7 months without a fit	3 or 4 min.	Matutinal	His "eyes keep moving," and he sees "colored lightsbefore a fit"	Frontal frequent	Rare	Dilated	Discs hyper... slight pulsating veins	R. ... L. ...
51	M	19	Has 3 sisters withfits, one of thesedied in a fit at 2d years of age, 2 sisters squint	14	Has 3 fits a week, formerly 1 fit daily	A few min.	Nocturnal	None	Frontal bad & frequent	Rare	Normal	Pulsating veins. L.E. int. strabismus & myopic crescent	Hr. counts ...m.
52	M	43	None	42	Has had many sensations, but only 3 fits	A few min.	Daytime	None	Occipital severeand frequent	None	Moderately contracted	Discs whitish, slight myopic crescents	R. ...– L. ...– 3
53	F	53	None	12	Has fits at variable periods, now about 1 a month, used to have attacks of fits	A few min.	Nocturnal	None	Fronto-temporal very severe and constant, occipital occasionally	Frequent & severe	Normal	—	R. H.m. L. H.m.
54	M	22	None	21	Has about 1 fit a month, occasionally 2 or 3 in rapid sequence	A few min.	Matutinal	None	None, occasionally frontalafter a fit	Matutinal daily	Moderately dilated	Pulsating veins. L.E. phys. cup.	R. ...let.– L. ...let.– L
55	M	28	None	22	Has about 3 sensations a month, even more, about 2 bad fits a year	A few min.	Matutinal, rarely at night	None unless a twisting to his right side	Frontal, occasionally occipital, both less than formerly	None	Normal	—	R. ... L. ...
56	F	17	Eldest brother has had 2 fits	2	Nearly daily, had no fits from 2 to 12 years of age	A few min.	Daytime	None	None	None	Moderately dilated	Pulsating veins. L.E. extr. strabis.	R. ... L. ...
57	M	28	None	9	Has 1 fit a fortnight	2 or 3 min.	Both in daytime and at night	A "rising to head, & then it twists round to the left"	Vertical, occasionally not marked	None, but has unsteady head feelings	Moderately dilated	Discs whitish, pulsating veins	R. ... L. ...

Vision and Refraction before Mydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
$\frac{6}{6}$ — 3 D. = $\frac{6}{12}$ $\frac{6}{6}$ — 2.50 D. = $\frac{6}{12}$ ii.	Homatropine	— 3 D. = $\frac{6}{12}$ — 2.50 D. = $\frac{6}{12}$	— 2.50 D. for constant wear	9 mos.	JULY—Has now about 1 fit in 3 months. Thinks they are stronger in character. AUGUST—About the same. SEPTEMBER—Has had 2 fits since last note
∫ let. H.m. .50 D. = $\frac{6}{9}$ ii. L ∫ let. H.m. 1 D. = $\frac{6}{9}$ ii.	G. A. S. grs. iv., ad ℥i. t.d. ut. 3 days	+ 1.50 D. = $\frac{6}{9}$ let. Do.	No glasses		
R. $\frac{6}{6}$ ii. n.i. L. $\frac{6}{12}$ iii. n.i.	Do. 7 days	$\frac{6}{12}$ + 2 D. $\frac{1}{2}$ = $\frac{6}{12}$ + — 2.50 D. / + 2.25 D. = $\frac{6}{9}$ $\frac{6}{60}$ + 2 D. = $\frac{6}{12}$ + — 4 D. / + 1.75 D. = $\frac{6}{9}$	+ 1.25 D. [C.W. —4.50D.cyl.ax. ⁄ 170° + 0.75 D. —5.50D.cyl.ax. \ 170°	6 mos.	JUNE—Remains *in statu quo*
$\frac{6}{6}$ — 3.50 D. = $\frac{6}{9}$ iv. $\frac{6}{6}$ — 3 D. = $\frac{6}{9}$	Homatropine	— 1 D. = $\frac{6}{9}$ let. — 1.50 D = $\frac{6}{9}$	— 1.50 D. C.W. — 2 D.		Has not worn glasses
R. $\frac{6}{9}$ n.i. L. $\frac{6}{9}$ n.i.	Do.	$\frac{6}{6}$ + 1 D. = $\frac{6}{9}$ $\frac{6}{12}$ + 1.50 D. = $\frac{6}{9}$	No glasses		Remains about the same
R. $\frac{6}{12}$ n.i. L. $\frac{6}{18}$ n.i.	G. A. S. grs. iv., ad ℥i. t.d. ut. 3 days	$\frac{6}{18}$ i. + + 2.50 D. / + 5 D. = + 2 D. / + 2.50 D. cyl. ax. | v. $\frac{6}{12}$ + + 1.50 D. / + 4.50 D. = + 1.50 D. / + 3 D. cyl. ax. | v.	Ordered less + 1 D. sph. for constant wear	6 mos.	JUNE—Has had 1 fit since glasses. SEPTEMBER—No fits (one in July?). Much better in health
R. $\frac{6}{9}$ let. L. $\frac{6}{9}$ let.	Do.	+ + 1 D. / + 1.50 D. = + 1 D. sph. / + .50 D. cyl. ax. | v. = $\frac{6}{9}$ let. + + 1.25 D. / + 1.75 D. = + 1.25 D. / + .50 D. cyl. ax. | v. = $\frac{6}{9}$	No glasses		
iv. nts fingers only n.	Do.	+ + .50 D. / + 1.50 D. = 0 / + 1 D. cyl. ax. | v. = $\frac{6}{9}$ iv. + — 12 D. / — 9 D. = — 12 D. sph. = $\frac{6}{6}$	— 1 D. sph. + 1 D. cyl. ax. | v. C.W. — 8 D.	8 mos.	JULY—Has only had 2 fits since glasses, which he cannot wear regularly. Is better. SEPTEMBER—Still continues better. ? 1 fit
$\frac{6}{6}$ — 1.50 D. = $\frac{6}{9}$ $\frac{6}{6}$ — 3 D. = $\frac{6}{9}$ iii.	Homatropine	$\frac{6}{12}$ — 1 D. = $\frac{6}{9}$ let. $\frac{6}{60}$ — 2.25 D. = $\frac{6}{9}$ let.	— .75 D. C.W. — 2 D.	8 mos.	SEPTEMBER—Has had no fits but occasional sensations
H.m. 7 D. = $\frac{6}{9}$ let. H.m. 8 D. = $\frac{6}{9}$ ii.	G. A. S. grs. iv., ad ℥i. t.d. ut. 3 days	+ 7.50 D. = $\frac{6}{9}$ + 8 D. = $\frac{6}{9}$ let.	Ordered and refused.	—	Has not worn glasses
let.—1.50 D. = $\frac{6}{9}$ ii. t.—1.50 D. = $\frac{6}{9}$ iv.	Do.	+ + .50 D. / + 2 D. = 0 / + 1.50 D. cyl. ax. | v. = $\frac{6}{9}$ + + 1.50 D. / + .50 D. = 0 / + 1 D. cyl. ax. | v. = $\frac{6}{9}$	Ordered č — .50 D. sph. for constant wear	8 mos.	JUNE—Has gone some weeks without fits, then had 3 at one time in sequence, and now has been 3 weeks without fit. JULY—No fits, no ceph. Vertigo much better. SEPTEMBER Do., do., do.
R. $\frac{6}{9}$ i. n.i. L. $\frac{6}{9}$ n.i.	Homatropine	$\frac{6}{9}$ n.i. + em. / + 1 D. = 0 / + 1 D. cyl. ax. | v. = $\frac{6}{9}$ iv. $\frac{6}{9}$ iv. + 1 D. = $\frac{6}{9}$ + + .50 D. / + 1.50 D. = + .50 D. / + 1 D. cyl. ax. | v. = $\frac{6}{9}$ iv.	Glasses refused.	—	Has not worn glasses
R. $\frac{6}{12}$ n.i. L. $\frac{6}{20}$ n.i.	G. A. S. grs. iv., t.d. ut. 3 days	+ — 1.50 D. / + 1 D. = — .25 D. / — 2.25 D. | 20° = $\frac{6}{9}$ iii. + — 1.75 D. / + 1.25 D. = 0 / — 2.25 D. cyl. ax. \ 120° = $\frac{6}{9}$ v.	— .75 D. — 2.25 D. cyl. ⁄ 20° — 1 D. — 2.25 D. cyl. ⁄ 120°	—	Has not worn glasses
R. $\frac{6}{6}$ n.i. L. $\frac{6}{18}$ n.i.	Do.	+ — 3 D. / + 3 D. = $\frac{6}{12}$ + — 3 D. / + 2.50 D. = — 6 D. cyl. ax. — / + 1.50 D. = $\frac{6}{9}$ — 6 D. cyl. ax. —	Ordered less + 1.50 D. sph. for constant wear	6 mos.	No fits while using atropine drops. JUNE—No fits. JULY—? 1 fit. Is much better in health

XUM

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when Fits began	Frequency, &c., of Fits.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fundus.	Vision at before
58	M	19	One brother had fits	18	Has about 1 fit in 3 weeks and sometimes attacks of many fits in rapid sequence	A few min.	Nocturnal and evening	Has post-orbital pains before a fit	Frontal frequent and post-ocular as aura	Occasionally before a fit not marked	Dilated, slow reaction	Phys. cup pulsation veins	R. $\frac{6}{9}$ L. $\frac{6}{9}$
59	F	32	None	7	Has about 1 fit a month, slight fits at 7 years, and decided fits at 11 years	10 min.	Matutinal	Twitching of right hand	Frontal and temporal frequent	None; unsteady feelings in head	Verydilated, slow reaction	Phys. cup pulsation veins	R. $\frac{6}{9}$ L. $\frac{6}{9}$
60	F	36	Mother had fits at 62 years of age	19	Has about 1 fit a month	10 min.	Matutinal	Her arms twitch for some days before a fit	Frontal not aching, but dull heavy sensations	Rare	Moderately dilated	Discs slightly hyper., pulsating veins	H.m. H.m.
61	F	14	None	11	Has about 6 fits in a day	About a min.	Nocturnal, also in daytime	Slight stiffness of mouth	Frontal severe	None	Dilated	Phys. cup pulsation veins	H.m. H.m.
62	M	16	None	13	Has 1 or more fits in a fortnight, when excited they are much more frequent	A few min.	Nocturnal, also in daytime	"Dazzling in eyes"	Frontal severe and frequent	Rare	Normal	—	Jet. H. Jet. H.
63	M	28	Grandfather had fits	20	Has 2 fits a month, formerly they were much more frequent	A few min.	Both in daytime and at night	Sensation in legs, has also "lightning flashes" before eyes	Fronto-temporal very severe and frequent	Rare, but has unsteady feelings in head often	Dilated	Discs slightly hyper., pulsating cup., pulsating veins	$\frac{6}{9}$ — $\frac{6}{9}$
64	M	22	None	5	Very frequent, daily	5 min.	Nocturnal	None; has seen "spots" before his eyes	Frontal, but only marked after a fit	None	Moderately dilated	Phys. cup pulsation veins	R. L.
65	F	32	None	30	At various periods, sometimes has 2 or three fits in rapid sequence	A few min.	Nocturnal	None	None	Frequent & severe	Moderately dilated	Normal; neal nebu	H.m. n.d.
66	F	52	None	23	At variable periods, occasionally with a 12 months' interval	A few min.	Daytime	Sees "hundreds and thousands of stars and colours"	Severe after a fit only; vertical	Frequent	Normal	Discs hyp pulsation veins. phys. cup.	R. $\frac{6}{9}$ L. $\frac{6}{9}$
67	F	20	None	4	Has often 8 fits a day. No fits between 4 and 7 years of age. Often attacks of several in rapid sequence	3 or 4 min.	Daytime, but occasionally nocturnal	"Lightning flashes and brilliant dots." Sensations in throat and eye	Fronto-temporal	None	Moderately dilated, slow reaction	Normal	R. L.
68	M	21	None	17	Has 2 or 3 fits a day. Has gone 4 months without a fit	Under ½ hour	Nocturnal, rarely in daytime	Frontal pains	Frontal frequent	Just before a fit	Dilated, slow reaction	Myopic cents, pulsating veins	$<\frac{6}{9}$ — $<\frac{6}{9}$
69	F	42	None	42	Began 2 months ago. Has had 2 fits (separate)	A few min.	Daytime	None	Occipital constant, vertical seldom	Rare	Moderately contracted	Phys. cup pulsation veins	H.m. H.m.
70	F	28	(1) One cousin, maternal; (2) one cousin, paternal; have fits	8	Has about 1 fit a week; no fits between 8 & 19 years of age. Sometimes 2 fits in 24 hours	10 min.	Daytime, rarely at night	None	Fronto-temporal, worse about time of fits	Rare, but has occasionally unsteady feelings	Moderately dilated	Discs slightly hyperaemic, pulsation veins, phys. cup.	R. L.
71	F	30	None	18	Daily; has laid 4 hours in succession of fits	A few min.	In daytime and at night	None	Frontal, worse after fits	Rarely	Moderately dilated	Discs hyperaemic, pulsation veins	H.m.
72	F	13	None	10	Has 10 or 12 fits a day. Once had 114 fits in a week. Has also uncounted fits at night	2 min.	Do.	None	Frontal and occipital, but not at all marked	Do.	Normal	Pulsation veins. phys. cup.	H.m. H.m.
73	F	12	Father when quite young	5	Has attacks of many fits in rapid sequence, about every 3 months	Depending upon no. of fits	Do.	Clutching of the left hand, and beating of heart	Frontal frequent	Do	Do.	Optic disc very white atrophic cupings. Borders very in colour	R. L.

XUM

Vision and Refraction before Mydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
$\frac{1}{4}$ – 2.25 D. = $\frac{2}{3}$ $\frac{1}{5}$ – 2.25 D. = $\frac{2}{3}$	G. A. S. grs. iv., b. d. ut. 3 days	$\frac{2}{3}$ let. + – 1 D. – 1 D. = $\frac{2}{3}$ – 1 D. – .50 D. cyl. ax. ⁄ 110° $\frac{2}{3}$ + – .75 D. – .75 D. = $\frac{2}{3}$ – 2 D. – 1.25 D. cyl. ax. ⁄ 75°	Ordered ċ – 1 D. sph. for constant wear	7 mos.	SEPTEMBER — No fits since glasses. Is perfectly well in health
R. $\frac{2}{3}$ let. n.i. L. $\frac{2}{3}$ let.	Do.	$\frac{2}{3}$ let. + + .50 D. + .75 D. $\frac{2}{3}$ let. + + .25 D. + .50 D.	No glasses.		
H.m. 1.50 D. = $\frac{2}{3}$ H.m. 2 D. = $\frac{2}{3}$	Do.	+ + 2 D. + 2.50 D. = $\frac{2}{3}$ + 2.50 D. + .50 D. cyl. ax. ⁄ v. + + 3.50 D. + 2.75 D. = $\frac{2}{3}$ + 2.75 D. + .75 D. cyl. ax. –	Ordered less + 1 D. sph. for constant wear	—	Has not worn glasses
H.m. 1 D. = $\frac{2}{3}$ iv. H.m. 1 D. = $\frac{2}{3}$ i.	Do.	+ 3.50 D. + 3.50 D. = $\frac{2}{3}$ let. + 4.50 D. = + 1 D. cyl. ax. ⁄ v. + 3 D. + 3 D. = $\frac{2}{3}$ ii. + 4 D. + 1 D. cyl. ax. ⁄ v.	Ordered less + 1 D. sph. for constant wear		
H.m. 1 D. = $\frac{2}{3}$ iv. H.m. 1 D. = $\frac{2}{3}$ i.	Do. 7 days	+ 1 D. = $\frac{2}{3}$ + 1 D. = $\frac{2}{3}$	No glasses.		
$\frac{1}{4}$ – 2.25 D. = $\frac{2}{3}$ $\frac{1}{5}$ Do.	Homatropine	– 1.50 D. = $\frac{2}{3}$ – 1.50 D. = $\frac{2}{3}$ let.	– 1.50 D. for constant wear	7 mos.	MAY—No fits since glasses. JULY—One fit after diving from a height into water. SEPTEMBER—No fits. Ceph. much better and no eye sensations
R. $\frac{2}{3}$ n.i. L. $\frac{2}{3}$ n.i.	G. A. S. grs. iv., ad $\frac{1}{3}$i. t. d. ut. 3 days	+ + .50 D. + .75 D. + + .50 D. + .75 D.	No glasses		
H.m. 1 D. = $\frac{2}{3}$ n.i.	Do.	< $\frac{2}{3}$ + 7 D. = $\frac{2}{3}$ < $\frac{2}{3}$ counts fingers n.i.	+ 6 D. C.W.		
R. $\frac{2}{3}$ let. n.i. L. $\frac{2}{3}$ let. n.i.	Homatropine	+ .50 D. = $\frac{2}{3}$ + .50 D. = $\frac{2}{3}$	+ 3 D. for near work	7 mos.	SEPTEMBER — No fits since glasses
R. $\frac{2}{3}$ let. L. $\frac{2}{3}$ let.	G. A. S. grs. iv. ad $\frac{1}{3}$i. t. d. ut. 3 days	$\frac{2}{3}$ + 2.50 D. = $\frac{2}{3}$ $\frac{2}{3}$ + 1 D. = $\frac{2}{3}$	+ 1 D. + .50 D. ⎫ for near work	7 mos.	JUNE—Says she is much better. AUGUST—Has had 20 fits. SEPTEMBER—4 fits since last visit. Says she has never been better.
< $\frac{2}{3}$ – 8 D. = $\frac{2}{3}$ < $\frac{2}{3}$ – 2 D. = $\frac{2}{3}$	Nil.	Did not reappear.	No glasses	—	Did not return
H.m. 3.50 D. = $\frac{2}{3}$ iv. H.m. 3 D. = $\frac{2}{3}$ let.	Homatropine	$\frac{2}{3}$ + 4.50 D. = $\frac{2}{3}$ let. < $\frac{2}{3}$ + 4 D. = $\frac{2}{3}$ let.	+ 5 D. + 4.50 D. ⎫ for near work	—	Did not reappear
R. $\frac{2}{3}$ let. L. $\frac{2}{3}$ let.	G. A. S. grs. iv. ad $\frac{1}{3}$i. t. d. ut. 3 days	$\frac{2}{3}$ + 1 D. = $\frac{2}{3}$ let. $\frac{2}{3}$ + 1 D. = $\frac{2}{3}$ let.	No glasses		
H.m. 1 D. = $\frac{2}{3}$ Do.	Do.	+ + 2 D. + 2.25 D. + 2.50 D. + + 3 D.	+ 1 D. for near work	6 mos.	JUNE—1 fit. Does not wear glasses always. SEPTEMBER—Does not wear glasses. Is *in statu quo*
H.m. 1 D. = $\frac{2}{3}$ H.m. 1 D. = $\frac{2}{3}$	Do.	$\frac{2}{3}$ + 2 D. = $\frac{2}{3}$ $\frac{2}{3}$ + 2 D. = $\frac{2}{3}$ let.	+ 1 D. for constant wear	Do.	Fits many less while using atropine drops. MAY—During day has had only 4 fits, since then some uncounted fits at night. Mother says "wonderfully better." SEPTEMBER—*In statu quo.* Falls so often she cannot wear glasses
R. $\frac{2}{3}$ n.i. L. $\frac{2}{3}$ n.i.	Do.	$\frac{2}{3}$ + + 4.75 D. + 4.50 D. = $\frac{2}{3}$ iii. + 5 D. + .50 D. cyl. ax. ⁄ v. $\frac{2}{3}$ + 4.25 D. + 4 D. = $\frac{2}{3}$ + 4.75 D. + .50 D. cyl. ax. ⁄ 120°	Ordered less + 1 D. sph. for constant wear	—	No subsequent note

XUM

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when Fits began	Frequency, &c., of Fits.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fundus, &c.	ion and efore
74	F	20	Sister had fits when suffering from chorea	20	Has about 1 fit a week	A few min.	Daytime	None	Frontal and vertical, marked after a fit	Very severe after a fit	Moderately dilated, slow reaction	Patient could not stand light in eye Hyperæthetic	R. L.
75	F	7	None	14 mos.	Has 3 fits daily	5 min.	In daytime and at night	None	Frontal frequent	None	Dilated, slow reaction	Pulsating veins, which are larger than normal compared with arteries	Cannot
76	F	39	Father and brother both had fits	32	Has about 3 fits yearly	10 min.	Daytime	Frontal pains	Only just before a fit	Frequent & severe	Moderately dilated	Normal	R. L.
77	F	67	None	62	Has 4 fits a month, occasionally 2 or 3 in a week	A few min.	Nocturnal	Feels ill and pains in chest	Not marked	Rarely near a fit	Normal	Discs hyperæmic, slightly pulsating veins	Let. Let.
78	M	7	None	2	Has about 1 fit in 3 months	Do.	Daytime	None	Frontal frequent and severe	None, says he feels unsteady sometimes	Dilated, slow reaction	Pulsating veins, physiol. cup.	R. L.
79	M	44	One brother has fits	39	Has about 6 fits in a year	10 min.	Nocturnal	Humming in head, cold feelings in right leg	Frontal constant and very severe	None, but unsteadiness	Normal	Pulsating veins, physiol. cup.	H.m
80	F	38	None	16	Has 2 or 3 fits in a fortnight, & then goes perhaps a month without; no fit between 18 and 32 years of age	5 min.	Daytime	Black specks dancing before the eyes. Has "lightning flashes" occasionally	Frontal constant and severe, occipital occasionally; both bad after a fit	Very frequent	Moderately dilated	Pulsating veins, physiol. cup. slight	R. L.
81	M	44	None	43	Has had 3 fits in all, but often has sensations as if fit were coming on	Under ½ hour	Do.	Commotion in head	Occipital severe and frequent	Rare	Do.	Discs slightly hyperæmic pulsating veins	Let. Let.
82	M	19	None	3	Has 1 fit a week. No fits between 3 & 14 years of age	14 min.	In daytime and at night	None	Vertical severe and frequent	Often	Dilated	Slight physiol. cup., pulsating veins	R. L.
83	M	46	None	18	Has from 1 fit a week to 1 a month	A few min.	Matutinal	None	Occipital severe & very frequent	Daily on rising	Contracted	Pulsating veins	H.m H.m
84	M	28	2 sisters have fits	17	Has had 12 fits in 10 years, but often has slight attacks	15 min.	Nocturnal, seldom in daytime	A feeling of blackness for some days	Frontal severe and frequent, vertical only when reading	Do.	Moderately dilated	Do.	R. H.m L.
85	M	12	One sister has fits	12	Has had 2 fits in 6 months	A few min.	Daytime	None	Frontal and occipital, the latter always after reading for a time	Rare	Moderately dilated, slow reaction	Normal	R. L.
86	F	11	None	1	Has 3 or 4 fits a day	Do.	In daytime and at night	None	Vertical frequent	None	Very dilated, slow reaction	Phys. cup pulsating veins (infantile paralysis on right side)	R. L.
87	F	21	None	6	Has about 2 fits a week	10 min.	Daytime, also at night	Peculiar feeling in stomach	Not marked, rarely post-ocular and frontal	Rare	Very dilated, slow reaction	Phys. cup pulsating veins	R. L.
88	F	10	None	7	Has 3 fits a day, rarely going a few days without an attack. Is idiotic	Momentary	In daytime, also at night	None	Vertical severe and occasional	? she stumbles often in her walk	Moderately dilated	Normal	Cannot
89	F	27	None	3	At variable periods from 3 or 4 a day to 2 a year	A few min.	Matutinal	Sees black just before a fit	Frontal and occipital severe and frequent	Frequent	Do.	Do.	Let. to R

XUM

Vision and Refraction before Mydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
R. $\frac{5}{6}$ L. $\frac{5}{12}$	G. A. S. grs. iv. ad ℥i. t. d. ut. 3 days	$<\frac{5}{6}$ — 1 D. = $\frac{5}{6}$ $<\frac{5}{6}$ — 1 D. = $\frac{5}{6}$	— 1.75 D. for constant wear	6 mos.	JULY—About the same. Aug. —1 slight fit since last here. SEPTEMBER—No fits, no ceph., no vertigo
Cannot read.	Do., grs. iiss. t. d. do.	R. + 1.50 D. + 1.75 D. L. Do.	No glasses		
R. $\frac{5}{6}$ let. L. $\frac{5}{6}$ let.	Homatropine	+ em. + 1.50 D. = — .50 D. + 1 D. cyl. ax. | v. = $\frac{5}{6}$ + em. + 2 D. = — .50 D. + 1.50 D. cyl. ax. | v.	Ordered ē — .75 D. sph. for constant wear	6 mos.	Has not worn glasses
let. H.m. 1 D. = $\frac{5}{6}$ let. Do.	Nil.		+ 4.50 D. for near work		
R. $\frac{5}{6}$ let. L. $\frac{5}{6}$ let.	G. A. S. grs. iiss. ad ℥i. t. d. ut. 7 days	+ 2.50 D. + 2.50 D. = $\frac{5}{6}$ Do. + 2.50 D. = $\frac{5}{6}$	+ 1.50 D. for constant wear	5 mos.	SEPTEMBER—No fits, no ceph. Mother says he is quite well
H.m. .50 D = $\frac{5}{6}$ Do.	Homatropine	+ 1.50 D. = $\frac{5}{6}$ + 1.50 D. = $\frac{5}{6}$	+ 1 D. for near work	—	No note
R. $\frac{5}{6}$ iii. n.i. L. $\frac{5}{6}$ iv. n.i.	Do.	$\frac{5}{12}$ + .50 D. = $\frac{5}{6}$ $\frac{5}{6}$ + .50 D. = $\frac{5}{6}$	No glasses		
let. H.m. .50 D. = $\frac{5}{6}$ let. let. Do.	G. A. S. grs. iv. ad t. d. ut. 3 days	$\frac{5}{12}$ let. + .75 D. + 1.50 D. = + .75 D. + .75 D. cyl. ax. | v. $\frac{5}{12}$ let.	Ordered for near work	5 mos.	SEPTEMBER—Has had 1 fit since glasses, but his general condition is worse
R. $\frac{5}{6}$ no H.m. L. $\frac{5}{6}$ no H.m.	Do.	+ 1.75 D. + 2.25 D. = + 1.75 D. + .50 D. cyl. ax. | v. = $\frac{5}{6}$ ii. + 1.75 D. + 2.25 D. Do.	+ 1 D. for near work	Do.	JUNE—No fits. SEPTEMBER—3 weeks ago 3 fits. Does not wear glasses always
H.m. 1 D. = $\frac{5}{6}$ H.m. 1 D. = $\frac{5}{6}$	Homatropine	+ em. + 1.75 D. = + .75 D. = $\frac{5}{6}$ Do. + 1.50 D. cyl. ax. | v. Do.	Ordered less sph. for constant wear	4 mos.	JULY—2 fits. SEPTEMBER—2 fits. Is better in health.
H.m. 1 D. = $\frac{5}{6}$ Do.	Do.	+ + 1.75 D. + 2.25 D. = + 1.75 D. + .50 D. cyl. ax. | v. = $\frac{5}{6}$ + + 2.25 D. + 3 D. = + 2.25 D. + .75 D. cyl. ax. | v.	Ordered less + 1 D. sph.	—	Does not wear glasses
R. $\frac{5}{6}$ let. L. $\frac{5}{6}$	G. A. S. grs. iiss. ad ℥i. t. d. ut. 3 days	+ + 1.50 D. + 1.75 D. Do. + 1.50 D. sph. = $\frac{5}{6}$ Do.	No glasses		
R. $\frac{5}{6}$ L. $\frac{5}{6}$	Do.	+ + 2.75 D. + 3.50 D. + + 2.25 D. + 2.75 D.	+ 2 D. sph. for constant wear	4 mos.	JUNE—No fits while using atropine drops. JULY—Has had 2 fits and 3 slight sensations
R. $\frac{5}{6}$ let. L. $\frac{5}{6}$ let.	Do., grs. iv. ad ℥i. do.	$\frac{5}{12}$ iii. + + .25 D. + .50 D. = — .75 D. + .25 D. cyl. ax. | v. = $\frac{5}{6}$ iii. $\frac{5}{12}$ ii. Do. Do. = $\frac{5}{6}$ Do. cyl. ax. / 100°	+ 1 D. sph. for near work	4 mos.	SEPTEMBER — No fits since glasses, but still has sensations
Cannot read.	Do., grs. iiss. a. d. ℥i. do.	+ em. + 1 D. = — .50 D. + 1 D. cyl. ax. | v. = good vision. + em. + 1.25 D. = — .50 D. + 1.25 D. cyl. ax. / 120° good vision	Ordered ē — .75 D. sph.	Do.	JULY—Mother says she is decidedly better. Fits are not so numerous. SEPTEMBER—Better, but still has fits.
let. no H.m. no H.m.	Homatropine	+ .50 D. = $\frac{5}{6}$ let. + .75 D. = $\frac{5}{6}$ let.	No glasses		

No. of Case.	Sex.	Age.	Family History as to Fits.	Age when Fits began	Frequency, &c., of Fits.	Duration of Fits.	Time when Fits occur.	Aura.	Cephalalgia.	Vertigo.	Pupils.	Fundus.	Vision and Refraction
90	F	8½	None	8½	Two days ago had 15 fits in rapid sequence	—	Daytime	None	None	None	Moderately dilated	Pulsation veins	R. L.
91	F	19	None	19	Several fits at various periods, also many sensations	A few min.	Matutinal	None	Frontal frequent, occasional vertical	Rare	Normal	Discs slightly hyperæmic, pulsation veins	R. L.
92	F	16	None	15	At variable periods, often many in rapid sequence; has gone a month without an attack	Do.	Daytime, but also at night	None	Vertical always & severe, temporal rare	Frequen	Do.	Pulsation veins	R. L.
93	F	13	None	4	Has about 30 fits a day, also some during night not counted. Has had 150 in 1 week	Do.	In daytime and at night	"Strange feelings inside"	Frontal severe & frequent	None	Moderately dilated, slow reaction	Do.	
94	F	30	None	15	Has 2 or 3 fits daily	15 min.	Do.	None	Do.	Rare	Moderately dilated	Do.	R. L.
95	M	37	None	11	Has 2 or 3 fits daily, with 2 or 3 very heavy ones weekly	A moment	Nocturnal	Peculiar feelings in head, and giddiness	Fronto-vertical	Frequent & before fits	Normal	Do.	R. $\frac{6}{6}$ L. $\frac{6}{6}$
96	M	40	None	36	Has 2 or 3 fits a day	Momentary	Do.	Peculiar sensations	Vertical and post-ocular, rarely occipital	Frequent	Do.	Do.	R. L.
97	F	24	None	12	Has about 1 fit a month; no fits between 12 and 21 years of age	10 min.	In daytime and at night	None	Frontal occasional & severe	Rare	Moderately dilated	R.E. myopic crescent, L.E. central phys. cup, Bothey... sating veins	R. $<\frac{6}{6}$ cannot L.
98	F	27	One brother has fits	27	Has had 3 fits during the last 2 months, but has had many sensations	Do.	Daytime	Peculiar feelings in stomach	Frontal parietal for weeks together	Frequent, matutinal	Do.	Phys. cup, slight pulsating veins	R. L.
99	M	19	None	18	Has from 2 to 9 attacks daily since 8 months old	Momentary	In daytime, also at night	None	Frontal rare now, formerly very frequent	Rare, after a fit occasionally	Do.	Pulsation veins	R. L.
100	M	16	One sister has fits	2	At variable periods, now 2 or more fits in a year	½ hour	—	None	Frontal severe and frequent, worse after a fit	Frequent & severe	Normal	Central phys. cup, pulsating veins	R. L.

Vision and Refraction before Mydriatic.	Mydriatic used.	Vision and Refraction after Mydriatic.	Description of Glasses Ordered.	Length of time Glasses were in use.	Condition since wearing Glasses.
R. $\frac{6}{x}$ n.i. L. $\frac{6}{12}$ n.i.	G. A. S. grs. iiss. a. d. ℥i. t. d. ut. 7 days	$\frac{6}{12}$ + + 3.25 D. = $\frac{6}{6}$ + 3 D. $\frac{6}{60}$ + + 3 D. + 3.25 D. + 3 D. = $\frac{6}{6}$	+ 2 D. for constant wear	4 mos.	JULY—No fits. SEPTEMBER—No fits.
R. $\frac{6}{6}$ let. n.i. L. $\frac{6}{6}$ let. n.i.	Do., grs. iv. ad ℥i. do.	$\frac{6}{6}$ + 1.75 D. = $\frac{6}{6}$ $\frac{6}{6}$ Do.	+ 1 D. for constant wear	Do.	SEPTEMBER — No fits. Ceph. much better, in fact rare
R. $\frac{6}{6}$ let. L. $\frac{6}{12}$	Do.	< $\frac{6}{60}$ + + 2.25 D. = + 1.75 D. = $\frac{6}{6}$ + 3.50 D. + 1.25 D. cyl. ax. ⌈ v. < $\frac{6}{60}$ + + 2.50 D. = + 1.75 D. = $\frac{6}{6}$ + 4 D. +1.50D.cyl.ax./120°	Ordered less + 1 D. sph. for constant wear	Do.	JUNE—Had no fits since using atropine drops, which have apparently stopped a series of fits. JULY—1 slight fit, no vertigo. Ceph. better. SEPTEMBER—3 fits and sensations. Says she is very much better in health
Do. R. $\frac{6}{12}$ L. $\frac{6}{12}$	Do., grs. iiss. ad ℥i. do.	< $\frac{6}{60}$ + + 2.50 D. = + 2.50 D. = $\frac{6}{6}$ + 5 D. + 2.50 D. cyl. ax. ⌈ v. < $\frac{6}{60}$ + + 3.50 D. + 3.50 D. = $\frac{6}{6}$ + 6 D. + 2.50 D. cyl. ax. ⌈ v.	Do.	—	? if this case is hystero-epilepsy
Do. R. $\frac{6}{x}$ n.i. L. $\frac{6}{36}$ n.i.	Homatropine	em. + − 1.75 D. + + 1 D. + .50 D.			
Do. L. $\frac{6}{12}$ − 2 D. = $\frac{6}{6}$ L. $\frac{6}{60}$ − 2 D. = $\frac{6}{6}$	Do.	+ − 1 D. = − 1.50 D. = $\frac{6}{6}$ − 2.25 D. − 1.25 D. cyl. ax./100° + − 2 D. − 2 D. = $\frac{6}{6}$ − 2 D. − .50 D. cyl. ax./80°	Ordered c̄ — .75 D. sph. for constant wear	—	No note
Do. R. $\frac{6}{6}$ iii. L. $\frac{6}{6}$ iv.	G. A. S. grs. iv. ad ℥i. t. d. ut. 3 days	$\frac{6}{6}$ + + 2.50 D. = + 1.75 D. = $\frac{6}{6}$ + 1.75 D. + .75 D. cyl. ax. — $\frac{6}{60}$ + + 2 D. + 2.25 D. = $\frac{6}{6}$	Do. for near work (his work forbids the constant wear)	3 mos.	Cannot wear glasses at his work. Remains *in statu quo*
< $\frac{6}{60}$ − 12 D. = $\frac{6}{18}$ cannot count fingers	Do.	< $\frac{6}{60}$ + − 12 D. = − 10 D. = $\frac{6}{12}$ − 10 D. − 2 D. cyl. ax. — + + 2 D. + 3.50 D. = sees no letters	Wears — 8 D. for constant wear		No note
R. $\frac{6}{6}$ let. L. $\frac{6}{6}$ let.	Do.	$\frac{6}{6}$ + .50 D. = $\frac{6}{6}$ + .50 D. cyl. ax. ⌋ v. $\frac{6}{12}$ + .50 D. = $\frac{6}{6}$ + .25 D. cyl. ax. ⌋ v.	+ 1 D. for near work		
R. $\frac{6}{6}$ L. $\frac{6}{6}$ let.	Do.	$\frac{6}{6}$ + 1.25 D. = $\frac{6}{6}$ $\frac{6}{6}$ + 1 D. = $\frac{6}{6}$	No glasses		
R. $\frac{6}{x}$ n.i. L. $\frac{6}{18}$ n.i.	Do.	+ + 1.50 D. = + 1.25 D. = $\frac{6}{6}$ i. + 1.25 D. + .25 D. cyl. ax. ⌈ v. + − .50 D. + 2.25 D. = $\frac{6}{12}$ + 2.25 D. − 2.50 D. cyl. ax./20°	Ordered less + 1 D. for constant wear	—	SEPTEMBER—Has not yet obtained glasses

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Clinical Cases.

A CASE OF HYSTERIA IN A BOY, CHARACTERIZED BY REGULARLY RECURRING ATTACKS OF LETHARGY: TREATMENT BY HYPNOTISM.[1]

BY THEODORE DILLER, M.D., PITTSBURG, U.S.A.

THE valuable paper by Professor Hitzig, "On attacks of Lethargy and on Hypnotic Suggestion," published in *Brain* (Spring-Summer, 1893), has moved me to relate the prominent features in a highly interesting and instructive case of regularly recurring lethargy in a negro boy, aged 14 years. The chief phenomena manifested in my case were very similar to those observed by Professor Hitzig in his case. Furthermore, my patient was, like his, treated by hypnotism, but with less degree of success; indeed it is questionable whether any real lasting benefit resulted from the treatment in my case. The case is similar to Professor Hitzig's in that it was of traumatic origin (symptoms following a blow on the head); it was characterised by regularly recurring periods of sleep (eight days duration); there was lessened metabolism during the lethargic periods; the patient was highly susceptible to hypnotism.

I regret that I am unable to furnish exact data as to bodily weight of patient and as to his excretion of urea during the attacks and intervals.

The patient was seen by me for the first time at the Mercy Hospital, last November, at the request of Dr. Brumbaugh, who afterwards kindly transferred him to my care. At that time he was in one of his "spells." He was profoundly apathetic; face expressionless; eyes open. He

[1] Paper read before the Pittsburg Academy of Medicine, January 15, 1894.

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could not be induced to talk or manifest the least interest in what was going on around him. A pinch or prick of the skin was unnoticed. My first impression was that it might be a case of stuporous insanity or of melancholia attonitia. But upon learning the history of the case and observing its subsequent developments, I became convinced that the phenomena manifested were those of hysteria.

History.—Charles Gibson, mulatto, aged 14. Up to the beginning of his present trouble he was regarded as an unusually bright and intelligent boy, but full of mischief and animal spirits. In 1889 he went to Washington, where he lived with a relative for three years, until September, 1892, when he returned to Pittsburg. Two years before his return here he met with an accident which marked the beginning of the disease from which he now suffers. He was engaged in a dispute with a companion, who, becoming angry, struck him on the head with a heavy iron rod, inflicting two wounds over the posterior part of the left parietal region, as witnessed by two scars close together, one-half and three-quarter inches long respectively. The boy, who was rendered unconscious by the blow, was carried to a place of retirement, where he recovered consciousness and was attended by a physician who sewed up and dressed the wounds. It was not thought at the time that a fracture had been produced. The lad apparently made a good recovery without sequelæ. But a few weeks after the injury he was taken with a spell of "mutism," preceded, however, for a few days by symptoms of giddiness. These spells occurred regularly once a month until a few weeks before I saw him, since which time they have occurred about every two weeks. The spells were characterised by complete apathy, and always lasted exactly eight days—never a longer or shorter period. During the attacks the boy could not be induced to talk or move, or in any way notice his surroundings. He passed his urine and fæces in bed. He would take no food, but drank some water during the attacks. Towards the end of the attacks the tongue and lips became dry, the teeth covered with sordes, and the breath very foul. The skin was cold, clammy, and the bodily weight evidently diminished. He states that he could remember much that was said to him while he was "under the spell." The beginning of the interval was always characterised by a returning desire for food, which, in a few days, amounted to a voracious appetite. For a few days he would eat an amount of food which would suffice for two able-bodied men.

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During the intervals the boy was full of spirits, exceedingly bright and intelligent, up to all sorts of tricks and mischief. His cousin states he is "full of the devil."

Present State.—On November 30, 1892, I carefully examined the lad during one of his intervals, while he was at his best. I found him to be an unusually bright, intelligent boy, with excellent manners (such as might have put many white boys to shame). A search for morbid sensory and motor phenomena was entirely fruitless. An examination of the head revealed the presence of the scars already referred to. They were not painful. No depression in the skull could be discovered.

December 3, I hypnotized him by the suggestive method, as employed by Professor Bernheim, of Nancy, with the greatest ease. I suggested that the medicine I was giving him (a placebo) would be of great benefit to him.

December 5, hypnotized him in the presence of Drs. Koenig, Stewart, Barham, and McKennan. Arms were placed in a number of awkward and unusual positions when it was suggested that they would remain in those positions until removed by me. The arms always were left, until I moved them, in the positions I placed them, and often remained so for a length of time, impossible to be persevered in by any voluntary efforts. I suggested that he should fight, pray, cry, &c., all of which suggestions were obeyed by characteristic movements and attitudes of the hands and arms. Any part of the body was readily rendered anæsthetic by suggestions, so that pin pricks or other painful stimuli were unnoticed. These suggestions were repeated at several subsequent sittings. I then suggested that he would not have his next spell until after Christmas (the next attack was due a few days before Christmas). I did this, reasoning that it would be easier to effect by suggestion, a postponement of the attack than to entirely prevent its supervention. If I was able to effect a postponement of attack, then, I reasoned, this demonstration of power would, at a subsequent time, lend greater weight to a suggestion that the attacks should never return.

December 20, hypnotized; suggestion of December 5 renewed. (The next day I left the city, and remained absent until December 28).

December 23. It was noticed that he was restless, uneasy and wanted to be very industrious at work about the house (always, according to his cousin, a sure sign of an approaching attack).

24th. Loud, noisy, incoherent, calling people by wrong names.

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25th. Apathetic; knew no one except his cousin.

26th and 27th. The same.

28th. I saw him. He was in a semi-stuporous condition, but knew me and called me by name. I administered an emetic of ipecac., which produced free emesis and seemed to waken him up somewhat.

30th. He called at my office, and, except that he was a little taciturn, seemed to be in his normal condition.

The duration of this attack was about five days; the shortest he had ever had. According to his cousin, it was less severe than any previous one, the lethargy being much less pronounced. The noisy prodroms were entirely new features. On the whole, the cousin pronounced the attack as very different from any he had previously had. As usual, he ate voraciously after this attack.

I again hypnotized him and suggested that he would never have another attack. I passed a very mild galvanic current through his head, from forehead to occiput, principally for its psychical effect. I usually repeated this at his subsequent visits.

January 9, 1893. Hypnotized and told he would never get another attack.

January 21. Called at office with cousin, in a semi-stupid condition. Hypnotized; suggested that he would be well on Monday (23rd).

January 23. Called at office with cousin, decidedly more stupid than at last visit. Movements sluggish; face expressionless; refuses to talk. Rather strong faradic current passed through back and shoulders, causing him to show lively discontent, as evidenced by movements and outcries. He had eaten nothing for two days—nor had he had a movement of the bowels during this period. I sent for a biscuit, which I forced him to eat under threat of applying the strong current to his back. I also administered a drop of croton oil.

January 24. Oil acted, producing several copious stools. Still very lethargic. Called at my office. Breath very offensive; sordes on teeth and lips. I forced him to eat a sandwich.

26th. Called at office alone; bright and talkative to-day; ate and drank freely.

It will be noted that this attack lasted five days. It was much less profound than usual. The boy, instead of remaining in bed throughout it, was, by my orders, kept up, and was compelled to eat twice, although the sum total of the food ingested was trifling.

On the whole both the attacks here described were shorter and less severe and characteristic than usual. The suggestion made before the first of these two attacks was a failure in that the attack came on before Christmas, when it was suggested that it would not come on till after Christmas. Yet its appearance was actually postponed a few days, and when it did come it was quite atypical, of shorter duration and less severe than usual. The suggestions made after the first of these attacks, that he would never have another spell, was largely a failure too; yet it resulted in making the attack which did come of three days less than the usual duration and by far the mildest he had ever had.

In both these attacks I noticed a lowered state of vital tone as shown in many ways. The skin was cold and clammy; there were sordes on tongue, lips and teeth; the breath was very offensive; there was an appreciable diminution in bodily weight; the pulse was slow and sluggish; the excretion of urine and fæces was markedly diminished.

I was greatly disappointed in not being able to continue my treatment and observations in the case, but I was prevented from doing so by a curious circumstance, which throws some light on the character of my little patient.

Soon after recovering from the second of the attacks described, he called at the police head-quarters in Pittsburg and told a pitiable story, relating that he had been lost in the city by his mother, who had travelled on to Washington. He told his tale so well that his story was readily credited, and he was furnished by the police department with a ticket to Washington. He then went home, secretly packed up a few effects and at once proceeded to Washington, and has since made his home with a relative in that city. The authorities at the police head-quarters were greatly astonished when they learned they had been duped, as they stated that the boy's earnestness of manner and the entire plausibility of his story had been such as to disarm suspicion in any one.

I suspect that the lad was moved to resort to this piece of stratagem through fear of further coercive measures in treatment. Moreover, he had heard some talk of an operation being performed on his head, and I have reason to believe that he dreaded this radical procedure would be executed.

The scars on the head were not painful. No hysterogenetic zones were discoverable. Yet the attacks of lethargy in themselves; the absence of all objective symptoms; the mannerisms and *morale* of the boy, together with his extreme susceptibility to

hypnotism; and lastly (and of great significance) the marked modification in character and duration of the two attacks brought about by hypnotic suggestion, indicated that the attacks were hysterical. Moreover, the masterly treatise on "Hysteria," by Gilles de la Tourette, reviewed in a recent number of BRAIN, seem to conclusively show that these sleep phenomena belong to the manifestations of hysteria. This view is confirmed by Prof. Hitzig's opinion, and to it I would add the feeble weight of my own approval.

With these meagre and unsatisfactory report and remarks I will conclude what I have to say, leaving you, gentlemen, to make any further conclusions or deductions, or to take issue with those I have made.

A CASE OF LEFT HOMONYMOUS HEMIANOPSIA.

BY JOHN TURNER, M.B. (ABERD.)

Senior Assistant Medical Officer, Essex County Asylum.

EMMA JOHNSON, aged 54, a house-painter's wife, was admitted on July 5, 1889. She had a paralytic stroke four or five years ago. Duration of insanity said to be three weeks. First attack: Facts indicating insanity on the admission paper were :—Unable to say where she is or what she has been doing. Memory for recent events a blank. Bit the hand of a nurse and attempted to strike one of the other inmates of the union.

On admission.—Stout, dark haired woman, complexion rather dark. Loss of power in left arm and leg, and sensibility to pricking is diminished in the limbs of this side but equal for both sides of face. Is in a state of mania, restless and excited, especially when talked to. Says her name is "anything." That she has sold her rings to get bread with. That she lives on her rent. She has no money and her husband does no work. Good health.

July 8.—She is very helpless on account of the paralysis of left arm and leg. The arm is contractured and semiflexed at elbow, and flexed at wrist and fingers ; left leg very slightly flexed at knee and fixed so that she cannot move it (anchylosed). She can lift both her legs off the bed, but with difficulty. Both plantar reflexes are absent, both knee jerks much exaggerated. Tendency to ankle clonus in left foot. Sensibility of left leg to pricking is almost *nil*, and much blunted and perverted in the right. Left leg cold, right warm. Both legs are marked extensively from the knees inclusive to feet with cicatrices of ulcers. Has some white cicatrices on her arms, especially the right, and the skin is darkened around. Chest healthy. She is very delusive ; lies and talks to her daughter, who she says is up above her and swears a great deal. Said she walked a mile yesterday. Is in bed.

September 20.—Her pupils are unequal, the right being the larger, and remained so till she died. They do not react to light,

and the right only accommodates for near objects. Tongue straight. When she smiles her mouth is drawn up on the left. *Analgesia:* Marked on both sides of face, chest, neck, arms, and legs. *Anæsthesia:* Some on the left side, none on the right upper extremity, none on either side of lower extremity that I can detect.

Localization of prick or touch.—Is generally seven or eight inches out on left side, less on the right (tested by telling her to place her fingers on the spot that I prick), same for legs and arms (tells correctly how many times she is touched or pricked on legs); *Mentally:* Is very talkative, boisterious spirits as a rule, delusion that her husband visits her every night. During 1890 no marked signs to note. As a rule is very cheerful and easily aroused to boisterous mirth. Sits quietly all day. Vomited one night in August.

March 20, 1891.—In a state of secondary dementia, dirty in habits and helpless. Grins and laughs when spoken to.

June 20.—She is stout and looking very well. Very talkative; will sit and talk to another patient who is not taking the slightest notice of her. Laughs frequently; excitable and irritable, and uses filthy language.

August 24.—She cannot walk without assistance. Protrudes tongue to left. She is quite unaware when I prick her on the left side of face or left hand, but calls out directly I do so on the right, or on either leg. She keeps chattering and laughing, but is very obstinate, and when asked to do anything replies as a rule "I shan't." Does not seem able to fix her attention on anything for long together. She does not appear deaf. She can see with either eye, will tell the time, grasp pennies held before her without any hesitation, and count them correctly. Always called the sovereign I was testing her with, half-a-sovereign. There is no doubt that the *left* half of each field of vision is blind, but not I think quite up to fixation line. It is impossible to test her with any great precision, as she will not fix her eyes on any object sufficiently long together, and when I held up a little piece of paper on the end of a pencil she took no notice of it, but she immediately grasped at a penny held on her right side, but took no notice of one on her left until it nearly approached the middle line.

August 28.—Tested again to-day for hemiopia. When she is talking to me and looking straight at me, if I hold up a half-crown on either side of her about three inches apart from one another, between her and me and equally far from the middle line, and ask her what she sees, she will reply "a shilling," and will put up her hand to catch hold of the one on her right. She does not perceive

the other till I move it past fixation point, and then she grasps it without hesitation. Each eye was tested separately with similar results.

In September she was confined to bed with erysipelas of face, started probably from an injury—a blow to eye. Vomited several times during this illness. Her temperature rose to 104° F., but in the course of a few days fell to normal again.

In December she fell out of bed and fractured her left clavicle, but seems to suffer no pain from the injury or examination.

March 10, 1892.—She ate a quantity of soap this morning and her lips are much swollen. She has done this once before. Sits talking and laughing to herself and other patients.

May 18.—Is confined to bed, where she lies huddled up on her left side. Her left foot and lower part of leg is swollen and the instep is ulcerated.

June 3.—Tested for hemiopia again to-day with similar results. Slight superficial sore on left buttock; speaks and knows what I say.

June 10.—Abscess formed over sacrum which has burst.

June 21.—Enormous hard brawny swelling of right parotid. Superficial sore about size of five shillings on left hip.

June 24.—To-day she died.

Post-mortem five hours after death.

Body.—Thin, rigor mortis present. *Sores*: Slight superficial one on left hip, and some very slight superficial abraded spots about inner aspect of left knee, right hip and right foot. Hard swelling of right parotid.

Atrophy of left arm and leg; there was about an inch difference in the circumference of arm, forearm, and thigh; no shortening.

Head.—Scalp thick.

Skull of average thickness and density, deeply grooved for blood vessels internally, no deeply marked pacchionian depressions.

Dura-mater firmly adherent to skull over the posterior part of the superior longitudinal sinus, which latter contained a little dark clot easily removed.

Arachnoid.—A bath of lymph having a jelly-like aspect was seen to cover the whole vertex beneath the arachnoid. There were deeper lakelets over the posterior end of the two lower frontal convolutions and lower end of ascending frontal on either side, and also over each superior parietal lobe, but deeper on the right. The arachnoid was opaque and thickened. Meninges not adherent to cortex.

Circle of Willis abnormal, there did not appear to be any posterior connecting vessel on the right side. The vessels were generally small and slightly atheromatous.

Cerebrum.—There was atrophy of the whole cerebrum, but more pronounced on the right, especially in the right præ-frontal lobe and the tips of both temporal lobes. A portion of the second occipital convolution on the right had a shrivelled appearance contrasting very markedly with the similar convolution on the left (see fig. 1). Brain firm and tough.

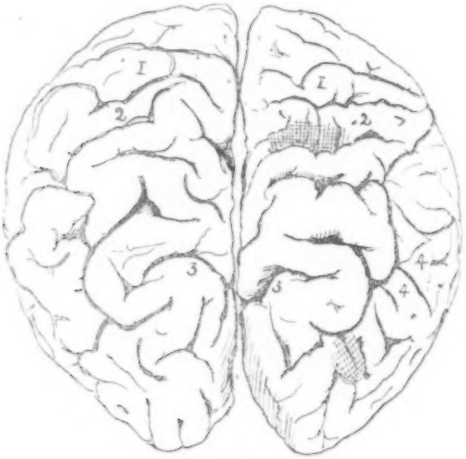


FIG. 1.

1. Is placed on ascending frontal convolution.
2. „ „ parietal „
3. Is adjacent to the parieto-occipital fissure.
4.4. Placed on angular gyrus.

The cross-shaded parts represent the seat of the lesion in the ascending parietal and adjacent superior parietal convolutions; and also the atrophied convolution in the second occipital gyrus.

The figure is outlined from a photograph taken of the brain.

Small cortical lesions in *right* hemisphere—old hæmorrhagic softenings.

(1) About the size of a pea in the back part of superior frontal.

(2) A rather large lesion implicating the upper part of ascending parietal and adjacent superior parietal lobe.

(3) A lesion in the second occipital convolution (see fig. 2). This lesion existed as a small irregular cyst situated at the junc-

tion of the cortex and white matter, and from it extended a degenerated tract running downwards and forwards beneath the posterior horn of the lateral ventricle for a distance of 6.5 centimetres. This tract occupied the lower most part of the optic radiations (see figs. 2 and 3).

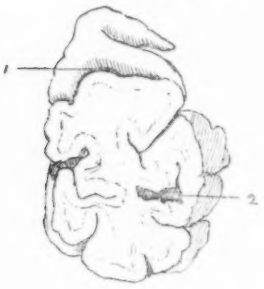


FIG. 2.

Horizontal section through parietal occipital fissure.
1. Parietal occipital fissure.
2. Small cyst.

FIG. 3.

Horizontal section about 1 cm. in advance of fig. 2 shows the posterior horn of lateral ventricle with small patch of degenerated tissue (black).

Both figures are outlined from photographs.

Lateral ventricles enormously distended with fluid. Small granulations on the lining membranes at the anterior ends; softened patch (old hæmorrhagic focus) observed on surface of *left* caudate nucleus about an inch from anterior end; this was seen on the floor of the ventricle.

Both optic thalami appeared shrunken but the right was the more atrophied of the two.

Transverse vertical sections made at distance of one centimetre from each other revealed numerous softened patches in the white matter of each præfrontal lobe and for a large extent below the right ascending parietal and right superior parietal lobes. Small softened patches also in right lenticular nucleus. The extent of the lesion in the occipital lobe has been referred to.

Right crus cerebri smaller than left. In the hardened preparation there was a difference of three millimetres in greatest length.

Right half of pons flattened, and the motor tracts on right side much smaller than on the left.

Right pyramid of medulla oblongata smaller than left.

Floor of fourth ventricle studded with coarse granulations.

No lesions observed in cerebellum.

Weight of fresh brain :—

Right cerebral hemisphere (stript) ...	313	grammes.
Left ,, ,, ,, ...	340	,,
Arachnoid and Pia	14	,,
Pons and Corpora Quadrigemina ..	21	,,
Cerebellum	106	,,
Total ...	794	,,

Eleven and a half ounces (327 cc.) of fluid collected from base beneath membranes and from ventricles.

Thorax:—Small patch of pneumonia at base of right lung. Heart weighed 227 grammes.

Abdomen.—Liver 1297 grammes, right kidney 113 grammes, left kidney 99 grammes.

This case is of interest, inasmuch as it presents a strictly limited and small lesion of a portion of the cortex and white matter in a region of the brain concerning whose function considerable difference of opinion still exists.

Briefly summarized there was with the small occipital lesion well-marked and persistent left homonymous hemianopsia, although neither the cuneus or the angular gyrus were involved.

Unfortunately the mental condition of the patient was such that a more precise account of the visual defects could not be obtained; it was impossible to accurately define the limits of the hemianopsia, although there was little doubt but that it reached up to fixation point, nor was it possible to test her for color hemiopia, &c.

Reviews and Notices of Books.

Lead Poisoning in its Acute and Chronic forms. Thomas Oliver, M.A., M.D., F.R.C.P.

Dr. Oliver has reproduced these very interesting lectures (Goulstonian, 1892) in book-form, thus contributing a valuable addition to the literature of this subject.

We confine ourselves in this notice to that part of his work which touches on the influence of lead on the nervous system. This, as may be expected, is manifested in a great variety of clinical forms. We will first notice the very serious acute nervous manifestation of plumbism known as lead encephalopathy.

After presenting the general symptoms of lead poisoning for a longer or shorter time, the patient passes into a condition easily taken for ordinary hysteria, so called "toxic hysteria"; this is soon followed by troubles of vision, amblyopia, temporary amaurosis. Convulsions, epileptoid in character, followed by fatal coma, may close the scene; or various mental troubles may supervene, such as mania or melancholia. There may be recovery with permanent or temporary loss of sight.

Dr. Oliver is of opinion that the severe pains of lead colic may be due to affection of the great sympathetic ganglia. This, however, must be purely hypothetical, from lack of facts either for or against, though the inequality of the pupils noted in so many cases is of interest in this connection.

A considerable space is devoted to the discussion of the chronic nervous symptoms of plumbism.

At the head of these come naturally the classic "wrist-drop." The occasional paralysis of the Duchenne-Erb group of muscles is also noticed.

Another form the chronic nervous affection may take, is one resembling progressive muscular atrophy of the Aran-Duchenne type and also of the leg or peroneal type.

An unusual form is described as "generalized" paralysis, in which large groups of muscles may be almost suddenly attacked; the disease may resemble an acute ascending paralysis with affection of limbs and trunk, but the head and neck are said to be usually spared. This form tends to recover, often rapidly. Death may occur from asphyxia.

The sensory symptoms of plumbism are not very striking. Dr. Oliver finds that pain often precedes the paralysis. Patches of anæsthesia and analgesia are fairly common on the arms and legs. Actual hyperæsthesia has been described.

The question as to whether the paralysis is of central or peripheral origin receives some attention. The myopathic theory was based on the assumption that the muscles contained the largest amount of lead. This assumption has been shown to be erroneous, and it has been found that the muscles, compared with other organs, contain the least quantity of lead, bulk for bulk.

The facts in support of the localisation of the seat of lesion in the grey matter of the cord is next discussed. V. Monakow found atrophy of the ganglion cells in the anterior lesions of the cervical cord. Oeller found capillary hæmorrhages, many of them involving the anterior cornual cells. The peripheral nerve trunks were also affected. Vulpian found myelitis after experimental lead poisoning.

These seem to be all the facts adduced in favour of the central hypothesis, and they seem to us too meagre to generalize from. The neuropathic theory of the causation of paralysis has scarcely received sufficient consideration in our opinion. The sensory symptoms mentioned as being common can scarcely be explained on any central hypothesis, and Dr. Oliver is careful to distinguish these symptoms from those of a hysterical nature.

Some interesting observations are made upon optic changes. He finds optic neuritis may be an early symptom in plumbism, and this quite apart from any kidney affection. There may be complete recovery from this, or the condition may progress to optic atrophy and total blindness.

Several interesting cases are quoted, with drawings of the discs, and pathological appearances after death.

Hysterical manifestations are occasionally met with, and may be prodromal, or may even be the only symptoms of the disease.

H. H. Tooth.

Brain Surgery, by ALLEN STARR, New York, 1893, 8vo, pp. 288.

THIS work, written by a physician who, although not himself an operator, has seen much cerebral surgery, aims rather at providing a judicial summary of our present knowledge than at originality or great minuteness of detail, but such a summary will be of no small value to those who have not the time or opportunity to study the numerous scattered records of the last few years. The scope of the book, its pleasant style, and even its faults—occasional dogmatism upon points which are still *sub judice*, and the enunciation of propositions without reference to exceptions—are those of a work, which will appeal less to the specialist than to him who requires only a practical working knowledge of the subject. We can indeed hardly explain the general character of "Brain Surgery" better than by quoting the following paragraph, which sufficiently indicates Dr. Starr's brevity and lucidity, accompanied by dogmatism :—

"The speech areas are of four kinds and in four locations. They are limited to the left hemisphere in right-handed persons and to the right hemisphere in left-handed persons. There is *the motor speech area* in the posterior part of the third frontal convolution, in which the movements concerned in the act of speech are controlled. The use of language and the power of talking are affected when this region is destroyed. There is *the auditory speech area* in the first and second temporal convolutions, in which the memories of word sounds are stored up. The understanding of language and the power of recollecting the names of objects are lost when this region is destroyed. There is *the visual speech area* in the lower parietal region, in which the memories of printed words are stored up. The understanding of written language and the power to read are lost when this region is destroyed. The *power of writing* is a part of speech and is usually lost when the motor speech area is destroyed, but its exact location is not fully determined; some cases pointing to the second frontal convolution, others to the lower parietal convolution near the hand centre as its probable cortical position." It should be added that the above is all that we find in the book on the subject of speech.

The first chapter of "Brain Surgery" deals mainly with the outlines of diagnosis, topography, and other preliminary subjects. Hare's method is preferred for the delimitation of the fissure of Rolando, and Reid's for the Sylvian fissure; but here as elsewhere, we miss the descriptions of alternatives, and there is no reference

to the work of Thane, Anderson and Makins, and others. It is, however, pointed out that great accuracy in the marking out of cerebral regions is not practically required, as the trephine opening is always necessarily sufficiently large to allow of some margin of error. The chapter concludes with the following, as the possible indications for operation:—"Operations may be performed for the relief of epilepsy, for the cure of imbecility, for the removal of clots, for the opening of abscesses, for the excision of tumours, for the relief of intracranial pressure—with or without drainage of the lateral ventricles—and for the cure of traumatic insanity." The usual traumatic conditions are not mentioned here or elsewhere in the book.

In the second chapter is an account of trephining for epilepsy, which deals mainly with post-traumatic conditions. The cases are divided into four groups, viz.:—1. Those in which the external evidences of injury and the focal symptoms point to the same region of the brain as being the seat of the lesion. 2. Those in which these do not coincide, it being then advised to operate in the region indicated by the nervous symptoms; two interesting cases are recorded to illustrate that the latter are a safer guide to the real site of the lesion than are the local signs. 3. Of operation in cases of general epilepsy, without focal symptoms, but of traumatic origin, the account is naturally most unfavourable; "occasionally the fits are relieved"; "the operation must be regarded as entirely exploratory" and "often nothing is found . . . and no result is obtained." This will be the experience of most surgeons, but more might well have been said upon the interesting question of the relief of epilepsy by all surgical procedures, *i.e.*, by operations *per se*, a relief which has given rise to so many vain hopes in the past. 4. Lastly, in cases of focal epilepsy in which no lesion is found after opening the meninges, the excision of the affected cortical area is not advised, chiefly upon the usual ground that such a procedure can only lead to further sclerotic changes. The calculated mortality after operations for epilepsy is taken from the most recent statistics to be seven per cent., and the general results are by no means favourable; of thirteen original cases only three are said to be cured, and these are reported only at intervals of two, three, and four months after the operation. And among the collected cases the longest period of immunity is eight months. "The question then arises, shall we continue to trephine any cases of traumatic or localised epilepsy? Judging by general results which are unfavourable, we should be inclined to stop. But when it is taken into consideration that in no case is it possible

without an operation to determine the exact pathological condition present, and that a certain proportion of the pathological conditions are removable, it is evident that an operation, if not attended with danger, may be reasonably undertaken. Some patients have undoubtedly been cured." To this summary we may add that a very full account is given of the changes found in several of the author's cases, and that Dr. Van Giesen contributes a careful description of their histological appearances.

The section upon "Imbecility due to Microcephalus" is very interesting at the present time, but deals with various congenital conditions besides that of microcephaly. The cases are divided into three main groups, the characters of which may, however, be combined, viz.: 1. Hemiplegia, with or without athetosis. 2. Mental defects of various grades. 3. Sensory defects of various types, especially deafness and varieties of visual defect. Epileptiform seizures are common in all the types, and the treatment of the organic epilepsies of childhood is also discussed in this place. It is pointed out that the clinical type depends rather upon the site than upon the pathological nature of the lesion, the latter including such diverse conditions as traumata at the time of birth, intra-uterine encephalitis and post-natal diseases of all kinds. The common factor in all of these affections is the production of sclerotic atrophy, and an analysis of 343 cases shows the following to be the lesions met with:—Porencephalus (132), sclerosis with disappearance of nerve elements (97), maldevelopment of the cerebral cells without gross defect (32), atrophy from embolism or thrombosis (23), meningo-encephalitis with adhesions, &c. (21), cysts external to the brain (14), hæmorrhages (18), hydrocephalus (5), and unilateral hydrocephalus (1). The primary origin of these various conditions, to which the writer has devoted much attention, is discussed, but without any fresh results being arrived at. As to the value of operation, it is clear that in the majority of the above lesions nothing is to be expected from trephining, but, in some cases of maldevelopment, it is suggested that "it is possible that anything which may stimulate latent powers of growth or may remove those influences which interfere with development, might result in improvement." It appears to us to be entirely problematical whether surgical intervention can produce these results. The actual statistics are, as might be expected, not good; thus 34 cases of craniotomy are analysed (Lannelongue's being excluded as wanting in precision of detail), and these yield 14 deaths—a much larger proportion than that of the cerebral operations upon adults; of the cases which survive the records are re-

garded as being too indefinite, or too recent to allow a satisfactory judgment as to the possibility of cure or great amelioration. It is remarkable that there is throughout the chapter only incidental references to the interesting and important question of premature synostosis, which is presumably one of "those influences which interfere with development," although the recent work of Bourneville and others proves conclusively that its importance has been enormously exaggerated.

The sections upon trephining for traumatic hæmorrhage contain nothing new to the surgeon; and operation is not advised in non-traumatic apoplexy. The cases of Championnière and Michaux are briefly related, together with an original case, in which operation revealed, not a clot, but a patch of softening. The main objection raised to this operation is the difficulty of diagnosis of the two latter conditions, but surely there are many cases in which no real difficulty is encountered in distinguishing hæmorrhage from embolism or thrombosis! No stress is laid upon what we take to be a far more serious difficulty, the radically defective condition of the cerebral vessels, and of the patient's general health.

The chapter upon abscess of the brain contains an excellent summary of our present knowledge, but makes no attempt to add to this.

In connection with tumours of the brain, Starr estimates that only seven per cent. are removable (tubercular masses being included), and he summarises the prospects of operation in the following paragraphs:—

"In any case which presents the general symptoms of brain tumour, and in which, during the progress of the disease, such local symptoms appear as indicate that the situation of the tumour is in or near the vertex of the convexity of the brain, the operation of trephining is indicated.

"This operation is not to be undertaken hastily in any case, as it is important to try the effect of anti-syphilitic treatment in those patients who may have had syphilis, and to watch for other symptoms or for signs of multiple tumours in patients who may have had tuberculosis. But if mercury and iodide of potassium fail to relieve the patient within three months, or if during that time the symptoms rapidly increase, the operation is not to be postponed.

"The chances of success are greatest in hard encapsulated sarcomata and fibromata, and in these cases the history will show little variability in the symptoms during the progress of the

disease. These tumours are usually on the surface and are easily removed. The chances of success are worth taking in glio-sarcomata and in soft infiltrating gliomata with or without cysts, as life may be prolonged by the operation and as several operations may be done successively if the tumour recurs. Such cases usually show much variability in the symptoms, as the tumours are vascular. The dangers are greater in their removal, partly from hæmorrhage, partly from inability to excise them entirely without great loss of brain tissue and consequent shock. It is not to be forgotten that these tumours are liable to recur. The chances of success are fairly good in cysts of the brain, provided the wall of the cyst is excised or the healing of the cyst from adhesion of its walls can be secured by permanent drainage. To empty a cyst and close the wound merely invites a refilling with fluid and is useless.

"Secondary carcinoma and sarcoma are not favourable (*sic*) for operation, as the chances of their recurrence are great, and the endurance of the patient is impaired by the primary disease."

To this long quotation we may add that among ninety-seven collected records of operations, there are forty-two in which the removal of the tumour was accomplished, and recovery ensued. Starr, in common with many recent writers, regards cerebral gummata as being generally amenable to medical treatment and as therefore not usually calling for operation, a view which our own experience certainly confirms. We miss in the chapter all reference to the puzzling cases in which focal symptoms are present but are misleading in their indications of the site of the tumour; and the very important question of the diagnosis of the *depth* of the tumour is dismissed with the remark that "the differentiation of cortical from sub-cortical tumours is still impossible," a remark which is certainly not universally applicable.

In a chapter upon trephining for hydrocephalus and for the relief of intracranial pressure, Keen's operation for tapping the ventricles is described, and seven cases are collected, of which the only one thoroughly successful is that of Broca, in which a hydrocephalic imbecile, with contracture of the right arm and epilepsy, was treated by drainage of the ventricles with very great improvement. The operation is recommended in such cases as being fairly safe and as likely to give a favourable result if the hydrocephalus be not secondary to other grave lesion.

On the subject of trephining for insanity, which has recently attracted a good deal of attention, the information is meagre. It is estimated that two per cent. of all cases are due to injury, and

that in such cases relief may be looked for. A few cases of this nature are mentioned, and it is concluded that "the number . . . is too small and the operation has been performed at a period too far removed from the trauma to warrant any definite conclusions." "It seems to me that in general paresis, a disease which has an organic lesion, a diffuse chronic meningo-encephalitis, it is impossible for the operation of trephining to do any good whatever."

By the above summary and quotations we hope to have indicated the general scope of "Brain Surgery," which is certainly a welcome addition to the literature of the subject, and in conclusion we may remark that this very impartial and fairly judicial work leaves the strong impression, which must have been growing steadily among those who have occasion to operate frequently, that at present, at least, cerebral surgery has proved less valuable than it at one time appeared likely to become, and that, unless the future brings considerable advances in our knowledge of pathology and diagnosis, this section of our art is little likely to advance in popularity.

WILLIAM THORBURN.

Études de Chirurgie médullaire. By A. CHIPAULT. Paris, Alcan, 1893, large 8vo, pp. 403.

THIS work is the first of a series, which is expected to extend to four similar volumes, and which is to embrace a full discussion of all questions connected with the surgical treatment of diseases and injuries of the spinal cord. Coming from the French surgeon who has especially devoted himself to this subject it is of great interest; and it contains by far the most complete account of the surgery of the spinal cord hitherto published. The extent of the recent developments of this branch of operative surgery may be gathered from the fact that over 500 cases are referred to, although it must be admitted that in a few of these, the operation has concerned the spine, and not the spinal cord.

Passing over a short historical introduction, we come to a description of the various operative procedures which have been adopted; and we may note in passing that the operation of opening the medullary canal is always spoken of as "lamnectomy," as suggested by Lloyd, a nomenclature, which although etymologically correct, is not likely in this country to oust the term "laminectomy" now generally employed. For this purpose the

central vertical incision is advised in all cases, and the bones are divided by rongeurs and forceps, and not by the trephine. A good deal of stress is laid upon the sub-periosteal method of resection, which is in the experience of the reviewer unnecessary, and it is stated that re-implantation of the bone after excision has not yet been practised; the latter has, however, been done in Manchester, but without any apparent benefit; and it would appear that the re-implantation of bone, either here or in the skull, is not of any great subsequent value, it being very doubtful whether the graft continues to live as bone. The author is opposed to the view of Prof. Horsley that the dura-mater should be opened in all cases, and his reasons for not doing so in many cases are certainly weighty. Much space is devoted to the question of suture of the pia-mater, but we think uselessly, as it is admitted that the operation is impracticable in cases of separation of the ends of the cord, and there is no reason to suppose that it would be of any service in cases of clean section; in fact, the disturbance involved, even in the careful hands of Dr. Chipault, would probably be productive of more injury than benefit.

In connection with the question of technique, a careful description is given of the author's method of attacking the anterior aspect of the medullary canal, by turning the cord aside, and thus removing tubercular granulations, and even chiselling away the protruding bodies of fractured vertebræ. Although much can be done by following up tubercular tissue to the front of the cord, and although the reviewer has frequently had occasion to do so, still we think that Dr. Chipault urges the matter too much; and that such an operation as the removal of the posterior edge of the body of a fractured vertebra through the medullary canal is not unlikely to be followed by considerable injury to the cord, from the necessary stretching thereof; nor, as a matter of fact, do the results attained by this method appear to have been more successful than those of laminectomy as generally practised. Much of the section relating to operative procedures is taken up with the discussion of methods which, aiming only at the treatment of tubercular lesions of the spine without reference to the cord, have no special interest for the neurologist, and these, although interesting in themselves, may therefore be here passed over. Other tentative procedures, such as wiring the vertebræ, are also referred to, and the latter appears to us to receive rather too flattering a reception. Quincke's method of spinal paracentesis is described, but the recent work of Ziemssen on this subject is not mentioned. (Possibly it was published after the book had gone to press.)

Coming to the detailed consideration of the treatment of injuries of the cord, we find little that is new; it is at once admitted that traumata of the cauda equina are, par excellence, the cases in which we are to expect good results, and the views expressed by the reviewer in 1887 are unmodified, except that we are advised not to delay operation for more than four weeks in cases which do not spontaneously recover. Equally certain is the small success obtained in injuries of the cord itself, and the failure is attributed largely to the operation having been undertaken too late, or to its not having been sufficiently radical; it is concluded from pathological study of the date of degeneration of the injured cord that little is to be hoped for where laminectomy is performed after the third day. It appears to us that insufficient weight is laid upon the inability of the cord to recover from destructive lesions of any kind, and that no reform in procedure is likely to overcome this difficulty. In one point of pathology Dr. Chipault differs from the reviewer, viz., in his conclusion that the majority of complete fractures of the bodies are followed by persistent compression, but here we cannot entirely agree with him, although the discussion of the question would require too much space for the compass of a review. Basing his conclusions upon this view, the reduction of displacements is advised as a substitute for laminectomy in injuries of the cervical region. Finally, at the conclusion of the section, is raised the interesting, and as yet unsolved question, how far are the cases reported as cured to be considered permanent recoveries, or how far do late secondary changes supervene?

With regard to gun-shot wounds, stabs, and other similar special lesions of the cord, there are some interesting collections of cases, but it is as yet too early to attempt generalisations.

The treatment of tubercular affections is next carefully considered, and the pathology of paraplegia in spinal caries is well discussed, without, however, providing any convincing solution of what still remains a somewhat open question; the main interest of the section lies in the enforcement of the author's method of attacking the bone disease through the medullary canal, already referred to and first enunciated in the *Revue de Chirurgie* for 1891. The general summary is as follows: But few cases of paraplegia due to tuberculosis anterior to the medullary canal are suitable for operation; the results have been unsatisfactory, and operation should be reserved for certain rare conditions, viz., paraplegia from pressure of cold abscesses; from direct compression by granulations; and from perimeningitis which has become scler-

osed. Successes have been obtained in other cases, but many of these would have recovered without operation.

Tumours are distinctly the most favourable field for spinal surgery, and here success has been great, most of these being extra-medullary, readily removable and non-recurrent; operation is altogether discouraged in the case of intra-medullary growths but on evidence which is as yet somewhat incomplete. Finally, a few rarer conditions which have led to laminectomy are discussed, but a remarkable omission is that of spina bifida occulta, which appears likely to give excellent results, and one case of which was reported by Mr. Jones, in the *British Medical Journal* for 1891.

The above remarks may appear to be of a critical, if not hypercritical nature, but we would not leave the reader with the impression that we do not regard this as an excellent work. It contains much careful reasoning, and has been prepared with unlimited industry by a master of the subject; and its study will be essential to those who wish to keep themselves abreast with this branch of surgery. There are numerous illustrations, which, although many of them are singularly inartistic, serve well the purpose for which they are intended; but it is remarkable that in two large coloured plates of laminectomies the incision represented is not that recommended by the author.

WILLIAM THORBURN.

Brain Diagrams on Slates. (DANIELSSEN & Co., Beaumont Street, London, W.)

MESSRS. DANIELSSEN & Co. have recently, at the suggestion of Dr. Whitwell, of Menston Asylum, made a set of engraved diagrams of the brain on slates for use in *post-mortem* and dissecting rooms. The diagrams are life size, twenty-five in number, and arranged on ten slates. The engraved outlines are filled in with white enamel, the Sylvian, Rolandic, and parieto-occipital fissures being, however, coloured red. Besides diagrams of the surfaces of the brain, pons, and cerebellum, there are numerous sections both vertical and horizontal ranging from the frontal region to the medulla oblongata. On the whole the details are clear and distinct but in some slates the outlines are very thick and the general appearance rather confused by the amount of detail. The slates seem to be well suited for use in the *post-mortem* room where the

site of any disease of the brain visible to the naked eye can be marked on the diagram in chalk at once by the pathologist even though his hands are soiled and wet. A rubbing can be taken from the diagram for addition to the *post-mortem* record, the site of the lesion being copied in ink or colour from the slate. In addition, however, to their use in the *post-mortem* room the slates seem well suited for use in the lecture theatre. The following complete list of the diagrams will show the thoroughness which has been aimed at in the preparation of these slates.

1.—Vertex. Base.

2.—Left lateral surface; right lateral surface.

3.—Inner surface of left hemisphere; inner surface of right hemisphere.

4 (after Dalton).—Horizontal section through anterior band and splenium of the corpus callosum; horizontal section through the anterior commissure.

5 (after Dalton).—Vertical section through the frontal lobes in front of the corpus callosum; vertical section through the corpus callosum, caudate and lenticular nuclei.

6 (after Dalton).—Vertical section through the corpus callosum, anterior pillars of the fornix and optic chiasma; vertical section through the corpus callosum, optic thalamus and crura cerebri.

7 (after Dalton).—Vertical section through the corpus callosum, and anterior pair of tubercula quadrigemina; vertical section through the lobulus quadratus and angular convolution; vertical section through the lobulus quadratus, cuneus and ends of the lateral ventricles.

8 (after Alexander Bruce).—Transverse section of the pons and cerebellum at the level of the sixth nerve; transverse section through the pons varolii at the level of the exit of the fifth nerve.

9 (after Alexander Bruce).—Transverse section of the medulla oblongata at the point where the direct cerebellar tract passes backwards to the restiform body; transverse section of the pons varolii immediately above the level of middle cerebellar peduncles; transverse section through the posterior corpora quadrigemina.

10.—Pons inferior view; pons superior view; pons lateral view; cerebellum superior surface; cerebellum inferior surface.

R. Percy Smith.

Abstract of papers upon unusual forms of Muscular Atrophy, in several members of one family. By J. A. Ormerod, M.D.

I. "Sur la Névrite interstitielle, hypertrophique, et progressive de l'enfance." Par MM. J. Dejerine et J. Sottar. (*Mémoires de la Société de Biologie*, série ix., tome v.)

The authors first draw attention to the form of muscular atrophy described by Charcot and Marie, Howard Tooth, and others, which is sometimes known as the "peroneal" or "leg" type of muscular atrophy, since it commences in the lower limbs, sometimes by the name proposed for it by Hoffman, viz., "progressive *neuritic* muscular atrophy," since it is thought by him to be caused by disease of the nerves rather than by disease of the muscle substance or of the spinal cord.

The present two cases, while resembling this form of muscular atrophy in certain important particulars, viz., in the primary localization and the course of the muscular atrophy, and in the existence of a family predisposition to the disease, yet present additional symptoms which, even during life, should mark them as distinct; and the elaborate investigation into the morbid anatomy of one case gives to the authors' observations an independent and additional value.

The cases are those of a brother and sister. Little of importance in the family history; father died at 45 from congestion of the lungs; mother alive and well, age 61 (her father was a drunkard and she was a natural child); nine children born to them; of whom five died young; while of the survivors two males are healthy, the remaining two are the patients under consideration.

Case 1 (authors' summary).—Muscular atrophy and sensory affection in a woman of 44. Onset in early life with deformity of the feet, for which tenotomy was practised at age of 6. Marked double club-foot (varus). Atrophy of leg and thigh muscles, most

marked at the periphery. Atrophy of upper limbs (Aran-Duchenne type) diminishing from below upwards. Excessive spinal curvature (kypho-scoliosis). Well marked sensory trouble, delay in transmission of sensation, diminishing from periphery to centre. Lightning pains. Inco-ordination distinct in upper limbs, less distinct in the lower limbs on account of the atrophy. Walking difficult and uncertain, but still possible. Romberg's symptom present. Slight choreiform movements of head and trunk. Patellar reflex, olecranon reflex, and plantar reflex absent. Pupils small, with very sluggish reflex to light. Nystagmus (dynamic). Fibrillary contractions of muscles of limbs and face. Sphincters and nutrition of skin not affected. Marked alteration of electrical contractility without reaction of degeneration.

Autopsy.—Hypertrophy of nerve trunks and spinal nerve roots, with alteration of the posterior columns of the cord. Histological examination—atrophy of the primitive fasciculus of the muscles, with slight steatosis and well marked sclerosis of vessels without any interstitial myositis. Hypertrophic interstitial neuritis of the nerve trunks diminishing slightly from below upwards, highly advanced in the muscular and cutaneous nerves. In the nerve roots the interstitial lesion, being less fully organised, exhibits the character of a primitive mono-tubular, interstitial neuritis. Similar lesions in the spinal ganglia. In the cord—lumbar region, sclerosis of Goll's columns and Burdach's columns; cervical region, Goll's columns highly sclerosed in their posterior part, columns of Burdach comparatively sound. No bacilli found in the nerves.

Case 1.—Fanny H. Age 45 at time of death. A small, cachectic woman, who had never suffered from any illness except the present paralysis. This began in early life. She began to walk at 3 years of age, and that badly; her feet were noticed to turn then. The spinal curvature did not exist then, but developed gradually later. Tenotomy was done at the age of 12; apparently she walked worse after it. She married at 32, and has now one healthy child (examined by the author) age 11, and has had three miscarriages. The weakness of the legs has gradually increased, and during the last fifteen years the affection has spread to the arms.

Condition at time of observation (1890).—In the lower limbs there is extreme muscular atrophy of legs and thighs, diminishing towards the root of the limbs. The feet exhibit much deformity, thus there is extreme varus, so that the patient walks on the scaphoid; exaggeration of the plantar arch, dorsiflexion of the

first phalanges of the toes, plantar flexion of the two last. No movements of the toes remain, and of the feet only a certain amount of abduction.

In the upper limbs there is marked muscular atrophy of the hands (thenar and interossei muscles), somewhat less of the forearms. Movements of the fingers remain, but are enfeebled. Muscles of shoulder and back badly developed.

Extreme spinal curvature, kypho-scoliosis, convexity to left, in upper part of spine.

Walking is only possible when she has her surgical boots on; without them indeed she can scarcely stand. There is a certain brusqueness of movement in the legs, but no pronounced ataxia. She can scarcely stand without supports. Romberg's sign is present in slight degree. The movements of the upper limbs exhibit a slight inco-ordination increased by closing the eyes. Tendon reactions, patellar and olecranon, absent; skin reflex from sole absent. No genito-urinary trouble.

As to sensation. In lower limbs, tactile sensibility is absent from the feet to the upper third of both legs, reappearing gradually above this level. Sensibility to pain is present, but in lower part of limbs transmission is delayed several seconds. Thermal sensibility is considerably impaired, and delayed in transmission. In upper limbs, sensibility to touch, temperature and pain is also affected. Sensibility is normal in face and trunk.

Lightning pains in left thigh for many years.

Slight "dynamic" nystagmus.

Re-examined in 1891 (November), when matters had got worse, with the following result:—

She cannot walk, and can only stand with support, and then loses equilibrium when the eyes are closed. There is some slight degree of paralysis in the lower part of the face as evidenced by a prominence and downward droop of the lower lip. Fibrillary contractions are clearly seen in the levators of the angles of the mouth, and also in the muscles of the shoulders and arms.

Eyes.—The right eye has been operated on for cataract. The left pupil is small, it contracts very slowly and imperfectly under light. Nystagmus when the eyes are moved to the side, none during rest. Fundus normal.

Inco-ordination of movement cannot be demonstrated in the lower limbs on account of the advanced paralysis, but in the upper limbs it is evident enough (having increased since the last observation), and it is intensified by closing the eyes. As regards muscular sense, she can, when the eyes are closed, imitate with

one arm any position in which the other has been placed, but cannot at once touch with her finger any part of her body that she is told to.

Electrical conditions.—Farado-contractility extinct in muscles of legs, thighs and hands, diminished in forearms. Galvano-contractility extinct or diminished in same degree as farado-contractility. No alteration of polar formula. Much diminution of electro-sensibility.

Cutaneous sensibility had undergone no change since the last observation.

Mentally the patient was somewhat feeble and emotional, though not so distinctly affected as her brother.

General condition bad: she had bronchitis and emphysema. She died in January, 1892, outside the hospital, and was buried; but the authors in their zeal obtained leave to exhume the body, and owing to the emaciation of the subject and the extreme coldness of the weather, it was found in good preservation.

The nerves of the cauda equina, both anterior and posterior roots, were found to be much enlarged (double their usual thickness), greyish in colour, smooth. The nerve roots of a higher level (dorsal and cervical) were slightly, but not strikingly, enlarged. The cord itself was normal, except that the posterior columns appeared rather smaller than usual, and somewhat reddish grey in colour.

The muscles of the lower limbs were much atrophied and of a reddish grey, in other parts salmon-coloured, yellow, or streaky and variegated. The most affected were the muscles of the feet and the muscles of the legs. In the leg muscles, the lower parts, viz., those nearest the tendon, were generally more degenerated than the upper.

The anterior tibial nerve was normal above the anterior tarsal ligament, underneath the ligament thinned and shiny, and below it swollen. The posterior tibial, and two plantar nerves were greatly swollen, and gelatinous in appearance. In the upper limbs the muscles of the forearms were thinned but otherwise normal. The intrinsic muscles of the hands, however, were highly atrophied. The median nerve was enlarged, firm, and gelatinous in appearance. The ulnar and radial nerves were much less affected.

An elaborate microscopical examination was made of the peripheral nerves, nerve-trunks, nerve-roots, spinal cord, and muscles. In the muscles (L. anterior tibial, L. flexor hallucis brevis, R. thenar muscles) it was difficult to find any muscular

tissues proper. A few strings of sarcous elements, enclosed in a copiously nucleated sarcolemma were seen, and also empty sheaths of sarcolemma; these were embedded in fatty tissue through which ran veins and arteries (the arterial coats being highly thickened) and degenerated nerves. There was no increase of connective tissue in the muscles.

In the cutaneous peripheral nerves of the leg no myeline remained, the nerve being reduced to a string of connective tissue; in those of the thigh, a few myeline tubes could be seen enwrapped in connective tissue. There appeared to be no Wallerian degeneration, but the myeline sheath had been gradually thinned and destroyed, and the axis cylinder in its turn exposed and destroyed, by the formation of connective tissue around the nervous parenchyma; this process beginning at the periphery of the limb and spreading upwards. The changes in the peripheral nerves of the muscles were exactly similar.

In the nerve-trunks were similar changes which might also be said, broadly speaking, to begin at the terminal parts and to spread with decreasing intensity up towards the roots. Thus in sections of the internal plantar nerve, there is some thickening of the epineurium, and very great thickening of the perineurium so that each nerve-fasciculus is surrounded by a thick ring of connective tissue; the endoneurium is also thickened, though in a less degree. In the nervous tubes themselves the myeline and the axis cylinder have disappeared, and are replaced by multi-nucleated connective tissue. In the nerves of the leg are similar changes, but the connective tissue is looser and less fibroid. In the sciatic trunk here and there a myeline sheath appears and within these (where visible) a normal axis cylinder.

Concerning the nerves of the upper limbs it need only be said that they showed the same lesions, but in a less advanced degree than those of the lower limbs.

A special description is given of the microscopical anatomy of the hypertrophied nerve-roots, as here the lesion was not so far advanced as to obscure its mode of development. The anterior roots were more highly diseased than the posterior. Each nerve tube was wrapped in a casing of loose newly-formed connective tissue composed of fusiform cells mostly parallel to the nervous axis. The myeline sheath was thinned in various degrees down to complete disappearance [but in no part broken up into blocks and balls, nor were the nuclei and protoplasm of the sarcolemma increased as in Wallerian degeneration], the axis cylinder was normal so long as there was any myeline left around it, and even

in some parts where it lay bare. Transverse sections showed connective tissue massed around areas which represented nerve tubes, and which in some cases showed remains of myeline, in others myeline and granular *débris*, the results of nerve disintegration.

The lower spinal roots were more diseased than the upper.

The authors regard the process as an interstitial neuritis, a proliferation of the connective tissue around the individual nerve fibres, commencing at the periphery of the nerves and spreading along their trunks up to the nerve roots. This connective tissue increase causes, in its early stages, enlargement of the structures as seen in the hypertrophy of the nerve roots, while in its later stages of organization and contraction it destroys by compression the nervous parenchyma, first the myeline sheath, then the axis cylinder ("neuritis interstitielle hypertrophique monotubulaire à marche ascendante").

Bacteriological examination of the nerve tissue threw no further light on the nature of the disease.

As regards the cord, the main feature was a degeneration of the posterior columns; which would appear from its distribution to be secondary to the lesions of the lower nerve roots.

At the second sacral segment, Burdach's columns were degenerated along the edge of the posterior cornua of each side. Lissaner's tract was degenerated, the posterior cornua somewhat thinned.

At the second lumbar segment, the degeneration of Burdach's column was more diffuse, and began to shift towards the middle line.

At the twelfth dorsal segment, the area of degeneration borders the middle line, extending at its posterior part only into the column of Burdach.

At the fifth dorsal segment, the posterior third of Goll's column is degenerated, Burdach's column showing only a slight deficiency of fibres here and there. This degeneration of the posterior part of Goll's column extended right up the cord.

A certain amount of disease was noticed in Clarke's columns. The cells of the anterior cornua differed but little from the normal, such alteration as there was being quite trivial in proportion to the disease of the nerve roots. The nerve fibres in their course through the cord to the roots were perfectly normal, the proliferation of connective tissue beginning immediately the roots left the cord. The degeneration within the cord appears to have been an ordinary secondary sclerosis; the neuroglia of the cord as a whole was not affected. A ganglion from a posterior nerve root was

examined. The proliferation of connective tissue involved it as it did the nerves, but had not progressed sufficiently to destroy the cells.

Case 2 (authors' summary).—Man, aged 34. Muscular atrophy associated with sensory affections and lightning pains, onset at 14 years of age. Commencement of atrophy in the leg muscles, later extension of it to the muscles of the hands. Muscles of the proximal parts of the limbs intact. Club feet (equinus). Atrophy of hand-muscles of the Aran-Duchenne type. Fibrillary contractions in the muscles of limbs and of face. Considerable alteration of faradic and galvanic contractility, but no reaction of degeneration. Curvature of spine (kypho-scoliosis). Ataxia of upper and lower limbs, inability to walk in the dark. Giving way of the legs. Romberg's sign. Nystagmus (dynamic). Pupils small, and of Argyll-Robertson type. Commencing atrophy of left optic disc. Deafness. Abolition of tendon reactions (at knee and elbow), and of skin reflex from sole. Considerable modification of the various modes of sensibility, diminishing from below upwards. Marked hypertrophy and induration of all such nerves of the limbs as are accessible to palpation. Severe lightning pains, sometimes causing the patient to fall down. Micturition, defæcation, and genital functions normal. No trophic lesions of skin. Syphilis at 24 years of age. Excess in alcohol. Mental degeneration.

Case 2.—Brother of last patient, age 34.

History.—Curvature of the spine had been noticed by his mother from the age of 4 or 5. While being treated for this with Sayre's jacket, at the age of 14, his paralytic symptoms began. He often fell when running, had some difficulty in using his hands. Writing became difficult; he had also to give up his work as compositor. His speech became peculiar. He had lightning pains in the limbs, particularly the lower limbs.

Unsteadiness of gait, especially when he first got up to walk, continued till the age of 21. He then fractured the right thigh (accidentally) twice running; and after this the motor troubles in the limbs increased so much that he had to give up work altogether.

Syphilis at the age of 21; some excess in alcohol from the age of 20 onwards.

Present state (February, 1890).—A somewhat short, anæmic man, with muscular atrophy of upper and lower limbs, and deformity of hands and feet, and some shortening of the right lower limb (due to the old fracture of the thigh).

Talipes of both feet, marked equinus with a slight degree of

varus, plantar arch much exaggerated, length of foot shortened, first phalanx of toes (especially the great toe) in dorsiflexion, the second and third in plantar flexion. Well marked atrophy of the thenar muscles, less of the interossei. Much atrophy of the leg muscles, particularly of the antero-external group. The tibialis anticus can still produce a slight dorsiflexion of the ankle and the calf muscles a little plantar flexion. Little movement is possible beyond this. The thigh muscles are not exactly atrophied, but are badly developed. Nevertheless, they appear tolerably strong. There are fibrillary contractions of the muscles.

He can only stand with the help of a stick and with his surgical boots on. Romberg's sign is well marked. He walks somewhat on his heels, and with a slight amount of jactitation; he does not walk straight, but swerving as if from cerebellar disease.

Muscular atrophy (of the Aran-Duchenne type) in both hands, involving the thenar and hypothenar muscles, and the interossei; the thenar muscles are completely paralysed, the interossei retain a small amount of power. Fibrillary contractions in the abductor longus pollicis (L.). Muscles of forearms poorly developed; epitrochlear group atrophied. Flexors of fingers and of hands enfeebled: the other forearm muscles retain their power. No definite atrophy of arm and shoulder muscles; those of neck, face, larynx, pharynx, palate and tongue are normal. The deformity of the spine consists in a kypho-scoliosis, concavity to left, in the mid-dorsal region.

Sensibility.—In lower limbs tactile sensibility is abolished from feet to knees; at knees it reappears, but with errors in localization and delay in transmission, and gradually improves up the thigh. Sensibility to pain and to heat and cold also altered, the localization thereof imperfect, and the transmission delayed. In upper limbs there are similar modifications of sensibility, but less marked. In the trunk sensibility to touch and pain diminished. On face and within mouth sensibility normal in all its modes. Muscular sense normal.

Tendon reactions at patella, and at elbow, absent. Plantar reflex not obtained.

In moving the hands towards a given object, slight swervings are made; these are much more marked when the eyes are shut. Similar inco-ordination is seen in the movement of the legs, but chiefly when the eyes are closed. Vision normal. Pupils slightly unequal, rather small in size, no reaction to light, normal reaction to accommodation. Nystagmus when the eyes are moved, but

not while they are at rest. Lightning pains in the lower limbs, worst when he walks, often so severe that he has to stop, or may even fall down. Speech slightly scanning. Though of fair intelligence, the patient shows some moral signs of mental degeneration.

An electrical examination (made February 17, 1890) showed that the atrophied muscles did not respond to faradism, though it appears that they reacted to a magneto-electric machine; to galvanism the leg muscles responded not at all, while the flexors of the forearm responded to 20 ma., but without alteration of polar formula.

Between the date of these first observations (1890), and the present time (March, 1893), there has been some alteration.

The muscular atrophy and the strength of the muscles remain much as before. The inco-ordination has increased, he cannot walk in the dusk; his legs often give way, sometimes without cause, sometimes when the lightning pains attack him. He can reproduce with one arm the attitude of the other, when his eyes are shut; but when told to touch a point of his body has to grope for it.

There are fibrillary contractions in the levators of the angle of the mouth, some drooping of the lower lip, and some difficulty of whistling.

A formerly existing deafness has steadily got worse.

An important point (specially examined for in view of the *post-mortem* data obtained from Case 1) is that all the nerves which can be felt—ulnar at elbow, musculo-spiral in its groove, median on inner aspect of arm, peroneal near head of fibula—are enlarged to double their usual size and very hard to touch. They are not, however, tender.

The type of disease to which clinically these cases appear at first sight to be most closely related, is (as stated at the commencement of the article) muscular atrophy of the peroneal type. So far, indeed, as the muscular atrophy is concerned the resemblance is very close indeed. In members of one family, at a comparatively early age, a chronic muscular atrophy begins in the legs, causing (or at least accompanied by) club-feet, and subsequently it spreads to the hands. The authors notice a small difference, viz., that in these cases the toes assume a claw-like position (dorsiflexion of the first phalanx, plantar flexion of the other phalanges), which we presume was due to a preponderance of atrophy in the interossei. More manifest differences, however, are seen when we consider other aspects of the cases than their

muscular atrophy. Thus, on the sensory side, there is marked affection of sensation in all its modes, whereas in peroneal atrophy affection of sensation is the exception; and as regards that rather vague function, the muscular sense, it is noted that, though the patients could (with eyes shut) reproduce with one arm the position in which the other was placed, yet when told to touch a given part of their limbs, toe, finger, elbow, &c. (the eyes being shut), they could only do so by fumbling and groping. Again, as to co-ordination, the patients were ataxic. This fact (so far as the gait was concerned) was in the first case masked by the paralysis; but in the second there were the following symptoms of ataxy, viz., jactitation of the legs in walking, swerving and lurching, inability to stand with the eyes shut (Romberg's symptom), or to walk in the dark, difficulty in turning or in beginning to walk, sudden giving way of the legs. In the upper limbs the ataxia showed itself by clumsiness in picking up objects, and erroneous projection of the finger in touching a point, errors which were increased by closing the eyes. Along with this ataxia existed two important symptoms whereby the disease is assimilated to typical locomotor ataxy rather than to muscular atrophy, viz., (1) lightning pains, (2) the condition of the pupils, which exhibited miosis and (in the second case at least) reflex irido-phlegia. Lastly (and in view of the relationship of the patients, the fact is particularly interesting), there were some symptoms which are more usual in Friedreich's ataxia than in ordinary tabes or in muscular atrophy, to wit, (1) nystagmus, (2) spinal curvature, (3) the peculiar deformity of the feet.

Clinically, then, the disease appears to be a sort of hybrid, in which appear the symptoms of peroneal muscular atrophy, of true tabes, and of Friedreich's disease. All the more interesting, therefore, is the account of the morbid anatomy. Of this we have already given a full abstract; but it is to be remarked that, in spite of the symptoms pointing to spinal disease, no spinal lesion was found beyond such as could be reasonably ascribed to secondary degeneration of the posterior nerve roots (degeneration of postero-lateral and of Lissauer's columns, degeneration of postero-median columns higher up), while the primary lesion consisted partly in a simple muscular atrophy, partly, and more particularly, in an hypertrophic interstitial neuritis, starting from the very periphery of the nerves and spreading right up to their roots.

The authors are able to quote but one similar case, viz., that published by MM. Gombault and Mallet under the title, "Un cas

de tabes ayant débuté dans l'enfance" (*Arch. de Médecine Experimentale*, 1889, p. 385). In this case also there was muscular atrophy, affection of sensibility, inco-ordination of movement, onset in infancy, and a similar hypertrophic neuritis of nerve and nerve roots, with posterior spinal sclerosis, was found *post-mortem*. It was regarded, however, as a case of tabes in early life with special complications.

The suggestion with which the paper concludes is worth quotation, viz., that in similar obscure cases of muscular atrophy, we should examine by palpation such nerves as are accessible to us. In the second case enlargement of the nerves was thus detected during life.

II.—"Ueber chronische spinale Muskelatrophie in Kindesalter auf familiärer Basis." Von Prof. J. HOFFMANN (Heidelberg). *Deutsche Zeitschrift für Nervenheilkunde.*

Some prefatory remarks are made upon the present classification of muscular atrophies, which divides them, according to the supposed primary seat of the lesion, into spinal, neural, and muscular types. This classification is based, partly upon anatomical, partly upon clinical data. Among the clinical characteristics, heredity has hitherto been considered to appertain only to the muscular type (*e.g.*, the progressive muscular dystrophy of Erb), or to the neural type (peroneal atrophy). But the following cases shew that a progressive muscular atrophy of spinal origin may also exhibit this character, attacking, as do the other two types, several members of the same family.

Case 1.—A girl of 4 years old. Up to $\frac{3}{4}$ of a year nothing abnormal had been noticed, except that she was very fat; she could sit up and move both her arms. Soon after this she gradually became unable to stand, sit in bed, or even turn over in bed though she still moved the feet and arms. The subcutaneous fat began to disappear, and revealed wasting of the muscles.

She was examined in the spring of 1891.

The limbs and trunk were wasted, the face pale, but not wasted. No sign of rickets, thyroid not to be felt, no demonstrable enlargement of thymus. There was paresis and wasting of many of the muscles of the neck and shoulders, of the upper arms, of the forearms; the intrinsic muscles of the hands were thin and flabby, though their motor power was more or less retained. The tendon reactions were absent, the mechanical irritability of the muscles diminished; the nerve trunks were not thickened nor tender. No fibrillary twitchings. Electrical reaction of degeneration demonstrated in the biceps.

In the trunk, much weakness of the muscles of back and of abdomen. Lordosis of the lumbo-sacral region.

In the lower limbs, the muscles of buttocks, flexors of thighs, and all the thigh muscles were much atrophied and palsied. Muscles of legs weak and atrophied. Movements of toes slow, but possible. Tendon reactions quite absent. Plantar reflex weak. No fibrillary twitchings.

The paralysis was throughout of the flaccid type, symmetrical on the two sides; there was nothing like hypertrophy or lipomatosis of muscle; no trophic changes; sensibility appeared to be normal. And, except for the signs of bronchitis, there was no other token of disease. The chest trouble increased during the winter, and the child died therefrom in March, 1892.

Post-mortem.—The body was much wasted, and there was very little rigor mortis. The spinal cord was small at the level of the lumbar enlargement, but otherwise normal. The anterior nerve roots, however, were highly atrophied.

The muscular degeneration was most strikingly seen in the quadriceps femoris, yet many other muscles were of a pale, yellowish red, instead of their normal colour. Much more subcutaneous fat remained than was expected.

Microscopical examination gave the following results: Cerebrum, cerebellum, pons, and medulla normal, excepting the lower sections of the medulla where the degeneration of motor cells, more evident in the spinal cord, began to shew itself.

Spinal Cord.—In sections from the upper cervical segments, there were only a few (at most five or six) normal cells in the anterior cornua, the rest were atrophic, their processes being small and scanty, their bodies small, their nucleoli indistinct. No disease of blood vessels nor proliferation of neuroglia. The anterior nerve-roots, external to the cord, shewed some signs of degeneration.

In the lateral and part of the anterior columns (apparently the pyramidal tracts), and in the parts of the lateral column between the pyramidal tracts and the anterior cornua—was a slight increase of neuroglia. The spinal accessory bundle was degenerated.

At the third cervical segment, the number of sound and of diseased anterior cornual cells was about equal.

In the cervical enlargement (which was normal in size) there were very few sound cells in the anterior cornua (only four or five in about thirty sections), there were many cell spaces empty of contents. As before, there was degeneration of the anterior

nerve roots outside the cord, and slight degeneration of them just within the pia mater. In the dorsal cord, similar changes but less striking. The alteration in the pyramidal tracts could be traced down to this level, though faintly.

The lumbar cord was shrunken; the degeneration of the anterior cornual cells was more marked here than at any other part. The anterior roots were degenerated almost up to the anterior horn; the pyramidal tracts were normal.

The disease of the anterior cornual cells (and of their nerve roots) thus extended through the whole length of the cord; it was symmetrical on both sides, and appeared to be degenerative, not inflammatory, in origin; since the neuroglia of the cornua, and their fine nerve network, was apparently normal.

Some bundles of the cauda equina were thin and pale.

Of the *nerves* that were examined, the anterior crural nerve shewed degeneration (nuclei increased in number, fibres broken up); so in a less degree did the peroneal nerve, still less the median and radial. The hypoglossal was normal. (This disease of the nerve trunks was less than would have been expected from the condition of the nerve roots.)

The changes in the *muscular tissue* were most marked in the quadriceps cruris. Here there was little muscle tissue left; the space that should have been occupied by a fibre was filled with aggregation of nuclei; while around the spaces lay the usual web of connective tissue. There was no fatty infiltration of this connective tissue. There were some muscle fibres visible in varying stages of atrophy, circular, not polygonal, in outline. There were hardly any hypertrophied muscle fibres; and no dichotomy, vacuolation, nor fatty degeneration of the muscle fibres. In the intramuscular nerves there were a considerable number of degenerating fibres.

Other muscles, *e.g.* (1) the muscles of the back, (2) the anterior tibial group, the deltoid and biceps brachii, (3) the intrinsic muscles of the hand, shewed degeneration, but in diminishing degree. The masseter and digastric, diaphragm, and cardiac muscles were normal.

Case 2.—Younger brother of Case 1. A boy of $2\frac{3}{4}$ years old at the time of first examination (November, 1891). Till three quarters of a year old he had been healthy, and had moved his limbs naturally, and could sit up. But he never learnt to stand or walk, became unable to sit up in his cradle, became strikingly fat, at the same time that he became weak and flabby.

A large, pale child; much fat in the subcutaneous tissue; the

muscles beneath it wasted and flabby; he cannot lift his head from the pillow, nor sit up unless propped.

There was paresis of the muscles of the neck and shoulder girdle (muscular atrophy was difficult to demonstrate in consequence of the fatness); in the arms, weakness and wasting of the flexor group, paresis of the triceps; in the forearms, wasting chiefly of the flexor group; in the hands, a certain amount of weakness and wasting of the intrinsic muscles.

Weakness of abdominal, weakness and wasting of back muscles. No lordosis.

The most marked paralysis was in the glutei and muscles of thigh, flexion of the hip was impossible; abduction barely possible; extension of the knee very imperfect. Much weakness and wasting of leg muscles, though some movements of the toes and ankles could still be made.

As in the preceding case, the muscular atrophy was diffuse, though masked by subcutaneous fat; the paralysis was flaccid in type, the tendon reactions were absent, there were no fibrillary tremors; electrically reaction of degeneration was found. There were no sensory abnormalities and no trophic disorders of the skin, and in all other respects than the muscular atrophy of the body and limbs, the child was healthy.

The child got gradually worse, a feeble cough developed itself, the subcutaneous fat diminished as the muscular atrophy progressed. (The observations are recorded till August of 1892, *i.e.*, for nearly a year.)

The family history of these patients stands as follows:—

Neither in the condition of the parents, nor in the history of their parents, was there anything appearing to bear on the origin of the disease.

Fifteen children had been born to their parents.

(1) Boy, healthy at first, but early in life he lost the use of arms and legs. Was very fat. Died at 11 months.

(2) Girl, died of diphtheria when nearly three years old; not paralysed.

(3) Boy, died at one month, of fits; was very stout, but no paralysis developed.

(4) Girl, when ¾-year old could stand, but subsequently lost power of standing. The paralysis, according to the parents, began in the trunk, and was accompanied by pain.

(5) Boy, now 11 years old and healthy.

(6) Boy, very fat; paralysis began in the trunk; had pains.

(7) Girl, died at 11 days, of fits.

(8) Boy, a seven months child, died an hour after birth.

(9) Boy, suffered from obesity and from the paralysis, also from pains.

(10) Girl, Case 1.

(11) Boy, died of fits, aged $1\frac{1}{2}$ weeks.

(12) Boy, Case 2.

(13) Boy, died of fits, aged $3\frac{1}{2}$ weeks.

(14) Boy, died of diarrhœa, aged three months.

(15) Baby, a month old, so far healthy.

Out of this large family, therefore, six may be disregarded, as not having reached the age at which the disease might be expected to declare itself; and of the remaining nine, six (four boys and two girls) had the disease; one (a boy) had what appeared to be an early symptom, while only two (one boy, one girl) escaped it.

The mother gave it as her experience that in those who died of the disease no *rigor mortis* appeared. She regarded the occurrence of obesity as a bad omen. Towards the end of the first year, and often after the children had learnt to walk and stand, the paralysis began in the trunk and legs, spreading afterwards to the arms.

By a curious coincidence two other cases from a second family came under the notice of the author about the same time.

Case 3.—Boy, age $\frac{3}{4}$ when first seen (Nov. 1891). A bright little child, exhibiting no sign of rickets or of other disease than that here described. The limbs had much subcutaneous fat, but their muscles were small and flabby.

There was weakness of the muscles of the head and neck, so that he could barely turn his head on the pillow or lift it up unaided. Almost complete paralysis of the extensors of the spine, abdominal muscles very weak. Paralysis and atrophy of the glutei and flexors of the hip; very little power in the muscles of the thigh, rather more in those of the leg. In the upper limbs, paresis of muscles of shoulder girdle and of upper arms; even the hand muscles were small and weak. No fibrillation, no rigidity, tendon reactions absent; reaction of degeneration, complete in the thigh-muscles, partial in the legs and upper limbs. Sensibility normal.

Early in 1892 persistent cough began, which became gradually feebler, and blocking of the air-passages commenced. The paralysis of the arms increased. The child died of bronchitis, March 29, 1892. No *post-mortem* was made.

This child had been healthy till five months old, able to move its limbs and to sit up well. About that age it had a prolonged shivering fit, about 14 days after which the mother noticed that it moved its limbs less easily than before; definite paralysis of the legs was made out some eight weeks later.

Case 4.—A boy (apparently not seen by the author), elder brother of Case 3.

He had been large and sturdy from birth; got his teeth early, could say some sentences and sing at a year old. But in the course of his first year he became gradually and unaccountably weak in the back and neck; never learnt to walk, and later began to lose power in his arms. He died of bronchitis æt. $1\frac{3}{4}$.

He had been seen when 14 months old by Professor Schultze, of Bonn, who had found paralysis with reaction of degeneration in the quadriceps crudis and tibialis anticus of both sides.

The history of this second family is as follows:—

Parents healthy in every way—they had six children.

(1) Boy, Case 4.

2. Boy, present age, 8 years.
3. Boy, ,, ,, 7 ,,
4. Girl, ,, ,, 5 ,,
5. Girl, ,, ,, 3 ,,

} all healthy.

(6) Boy, Case 3.

Upon these cases the author bases a general sketch of the disease.

Beyond the fact that it appears to run in families, nothing is known of the causation of the disease. The parents, ancestry, and unaffected brothers and sisters appear to be on the whole healthy. So do the victims of the disease during the first few months of their life. Usually in the second six months of life the onset takes place, in a subacute or still more gradual fashion. The natural kicking movements of the legs become weak and hampered, presumably from affection of the muscles of the hip and thigh, and the muscles of the back become weak; so that, if the child has already learnt to stand or sit up, it loses the power of so doing. Thus it remains for a variable time. Then paralysis begins in the muscles of the head and neck, shoulder and upper arm; it can barely turn its head in bed, and has difficulty in lifting its arms. The muscles of the leg (below the knee) are involved about the same time, and the weakness spreads through in less degree to those of the forearm and hand. With this paralysis is associated atrophy of the affected muscles. At the

first this may be hidden, from the eye at any rate, by an abnormal amount of fat in the subcutaneous connective tissue; but this may disappear, revealing the muscular atrophy in all its severity.

There are neither cerebral nor bulbar symptoms, nor are the sphincters affected; there is no sensory affection; pains appear to be the exception. The paralysis is symmetrical, and progresses; so that death usually results before the fourth year of life from secondary pulmonary affections.

The order in which the muscles are affected appears to be (1) Muscles of back, of buttocks, flexors of hip, thigh muscles. (2) Muscles of head and neck, of shoulder-girdle and upper arm, of leg. (3) Intrinsic muscles of hands, and probably of toes.

The muscles are not rigid, but lax, and waste progressively; the tendon reactions are abolished; there is electrical reaction of degeneration; there are no fibrillary or fascicular twitchings. The muscles are not tender to touch, the nerve trunks are not tender nor enlarged. There is no hypertrophy of the muscles.

Post-mortem there is found atrophy or disappearance of the bulk of the anterior cornual cells throughout the whole cord, but most marked in the cervical and lumbar enlargements, and particularly in the lumbar. Atrophy of the anterior nerve-roots (at least externally to the cord), degeneration of the nerves (motor or mixed), and atrophy of the muscles supplied by them. The muscular atrophy is characterised by increase of the nuclei, the muscle tissue disappearing by degrees, without losing its striation, and without such changes as dichotomy of the fibre, hypertrophy of the fibre, vacuolation, or fatty infiltration. The affection of the nerves and muscles is to be regarded as secondary to the disease of the anterior cornua.

The diagnosis has to be made from infantile paralysis, (spinal and cerebral), peroneal muscular atrophy, progressive muscular dystrophy of Erb (pseudo-hypertrophic paralysis, infantile and juvenile muscular atrophy), the hereditary muscular atrophy of Leyden and Möbivs. It is distinct from all these diseases, inasmuch as it consists in a chronic degeneration of the anterior cornual cells, but, unlike most varieties of this disease (chronic anterior poliomyelitis, amyotrophic lateral sclerosis, progressive muscular atrophy of Aran-Duchenne type), it is a disease of infancy and runs in families.

The author has been able to find only one set of similar cases, viz., those published by Werdnig in the *Archiv. für Psychiatrie*,

vol. xxii., p. 437. The abstractor wishes to draw attention here to "a case of progressive muscular atrophy in a child, with a spinal lesion," published by Drs. Thomson and Bruce in the *Edinburgh Hospital Reports*, vol. 1. This was an isolated case, there being no other cases in the family.

III.—"Ueber eine hereditäre Form der progressiven spinalen mit Bulbär-paralyse complicirten Muskel-atrophie." Von Professor Dr. M. BERNHARDT. (*Archiv. für pathologische Anatomie und Physiologie und für Klinische Medicin. Band* 115, *Hft.* 2, *p.* 197.

Case 1.—P., a large, somewhat powerful-built man, aged 40 (January, 1882), complaining of inability to use the right hand properly in writing. He occasionally also stumbled with the right leg.

In February he noticed a sort of choking sensation in the throat whenever he laughed. The speech had now become somewhat thick, low, and occasionally hurried. The tongue appeared normal, also the lips, face, ocular and pupillary movements.

September, 1882. Well-marked bulbar paralysis. Speech low, toneless, difficult to understand. Tongue partially atrophic, cannot be protruded beyond the teeth, its farado-contractility diminished. Labials badly pronounced; difficulty in holding a cigar in the lips. Fibrillary twitchings in muscles of lips and chin. Farado-contractility of these muscles normal. Chewing and swallowing difficult. Cannot now write at all. Has a peculiar hopping movement in going up stairs. Well-marked patellae tendon reflex.

The bulbar paralysis continued to increase in April and May, 1883, and the patient died in August of that year.

Case 2 (first cousin once removed to Case 1, L. being grandson to P.'s aunt). October, 1882. L., a man about 32 years of age, who complained of weakness of the arms and neck. His head had a peculiar pose, falling forward as a rule with the chin upon the chest; the patient could only lift it either by his hand, or by a swinging movement executed with his whole body. There was atrophy and paralysis of the neck and shoulder-muscles, to wit, the trapezii, sternomastoids, levators anguli scapulæ, supra-spinati, and deltoids. Movements of the forearms and fingers were possible, but enfeebled. Faradic contractility was diminished in the muscles of the arm, forearm and hand, and was barely perceptible in the muscles of the neck and shoulder. No reaction of degeneration could be made out with the constant current.

The speech, face, tongue, lower limbs and sphincters were not affected. Tendon reactions normal.

Half a year later the only additional symptom was an occasional difficulty in swallowing.

The patient then passed into other hands, but the author heard from his sister that long before his death (which took place in 1884, two years after he was first seen) he could not feed himself, and the physician who last attended him regarded the case as one of bulbar paralysis.

Case 3 (first cousin to Case 2).—B., a man of 36 or 37, seen once only, March 1, 1888.

Much mental and bodily exertion during the last two years. Three-quarters of a year ago a sensation began of tension and weakness in the muscles of the neck and throat, particularly on the left side, radiating towards the left shoulder. Paræsthesiæ in the left cheek, difficulty and pain in chewing food, trouble in swallowing, spasmodic cough at night, with some spasm of the glottis. Then difficulty in balancing his head, which would fall forward or to the side.

On examination the patient was thin generally, but there was marked atrophy of the trapezii, splenii, supra- and infra-spinati, deltoids; with great diminution of their farado-contractility, but no reaction of degeneration. Muscles of the arms, forearms and hands thin, but not distinctly atrophied. Movements of arms at shoulder-joint deficient.

The head could only be moved feebly and tended to sink forward, chin upon chest.

No fibrillary tremors.

Dyspnœa upon the least exertion.

Tongue could be moved, but was thin, pointed, and showed furrows of atrophy. Swallowing difficult, singing impossible.

Soft palate moved normally; lips moved normally, but speech was difficult.

Upper facial muscles normal.

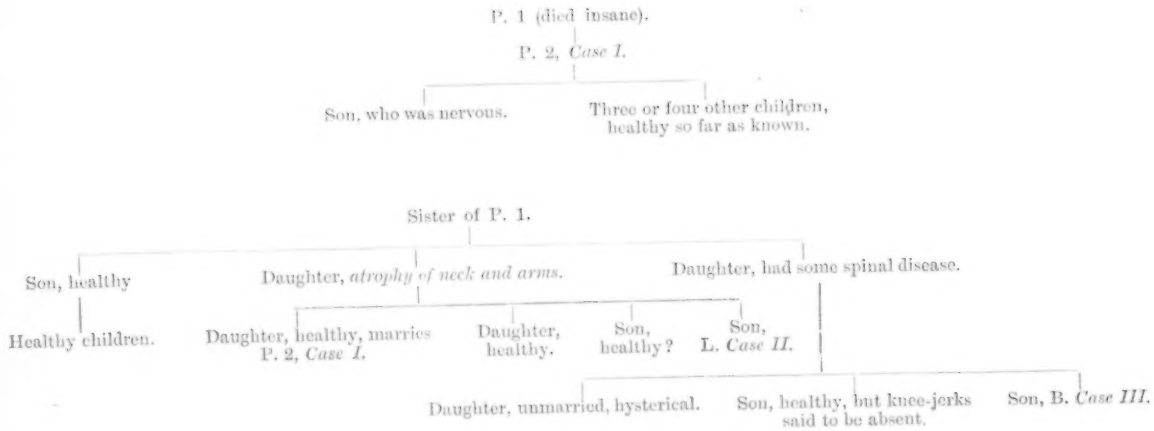
Reaction of tongue and facial muscles to electricity normal.

Lower limbs normal.

The patient died three months later.

The mother of Case 2 appears to have had the disease; according to her medical attendant she had wasting and palsy of the muscles of the neck and arm, and had to be fed.

The author evidently considers (though he admits that without a *post-mortem* rigid proof is wanting) that this affection is due to disease of the spinal and bulbar motor cells (myelopathic atrophy). It differs from myopathic atrophy (dystrophia muscularis progressive of Erb) in the early affection of the deep


P. 1 (died insane).
P. 2, *Case I.*
Son, who was nervous.
Three or four other children, healthy so far as known.
Sister of P. 1.
Son, healthy
Daughter, *atrophy of neck and arms.*
Daughter, had some spinal disease.
Healthy children.
Daughter, healthy, marries P. 2, *Case I.*
Daughter, healthy.
Son, healthy?
Son, L. *Case II.*
Daughter, unmarried, hysterical.
Son, healthy, but knee-jerks said to be absent.
Son, B. *Case III.*

muscles of the neck, in the speedy spread to the bulbar nuclei, and the (consequently) rapidly fatal course.

From the known types of myelopathic atrophy, however, it differs in the prominent part which heredity appears to take in its production, for it is generally held that progressive muscular atrophy of spinal origin does not run in families.

The hereditary nature of the affection is best illustrated by quoting the family tree given by the author.

From this it appears that nothing is known of the originators of this diseased stock (father and mother of P. 1); that in the second generation from them the disease appeared in two or perhaps three cousins; and, in the third generation, again in two cousins; lastly, it may be noticed that P. 2, a victim of the disease, intermarries with the daughter of his (diseased) cousin. Considerable interest, therefore, would attach to the children of this couple, but so far, at least, the disease had not appeared in them. P. 2 (Case 1) must have obtained the liability to the disease through his father, the others had it through descent in the female line.

Groenow on Double-sided Hemianopsia of Central Origin. (*Archiv f. Pysch.* xxiii., p. 339.) The first case of this kind has been described by Foerster. The author records a second, and refers also to a patient of Charcot's, who presented a similar loss of sense of locality. He argues that here also a double hemianopsia must have existed.[1]

Concentric narrowing of the half fields of vision in lateral hemianopsia is, as a rule, not to be regarded as depending on a primary disease of both visual centres. Hemianopsia, which may be referred to double-sided brain disease, is more probably indicated when the boundary between the defective and sound parts of the visual field considerably encroaches in places towards the active side (*e.g.*, as when beyond total loss of left half fields of vision of both eyes a partial defect occurs in the right). For a case to be absolutely typical, it is requisite that the halves of the field of vision supplied by one optic tract should be impaired in a first attack, and the halves supplied by the other tract in a second. These considerations are fulfilled by the following case:—

[1] Charcot's case will be found described on p. 152, *et seq.*, of N.S.S. translation of Charcot's Works, vol. iii.

A sculptor, Professor X., aged 58, had on 10th January, 1889, an attack of left-sided hemiplegia; the arm and leg were slightly paralysed, but he was able to use them. There was no loss of consciousness, but he noticed on resuming work a disturbance of sight, for which at the end of April, 1889, he consulted a doctor. Complete left-sided hemianopsia was diagnosed, with normal sharpness of vision. Ten months later he had another apoplectiform seizure, when he became forgetful, languid and sleepy. He slept for nearly two days, but could be roused without difficulty for his meals. Three days after this last attack it was found that he had lost the power of finding his way about; he believed himself to be in a strange dwelling, and demanded to be taken home; otherwise he seemed clear in his mind. His sight was disturbed, and though there was no absolute amaurosis, yet he could not, *e.g.*, recognise some men at work opposite his house. His field of vision was found to be extraordinarily restricted. Sharpness of vision in certain positions of head was good. He gradually improved so that he could find his way about his room alone. December 15th, Dr. Groenow first saw him. Smell, taste and hearing unaffected by attacks. Sense of position of limbs in space normal. Slight impairment of strength in left hand, sensibility to pricking normal. Sharpness of sight good; movements of eyeball free; no abnormal ophthalmoscopic appearances. The field of vision of each eye, however, is reduced to an irregular figure of 8, obliquely placed; the right lower and left upper quadrants in each eye being defective, though not absolutely blind. Coloured objects 5 mm. square were correctly recognised only in a limited central region scarcely reaching two degrees from the perpendicular through fixation point to the left, but nearly ten to the right upper. It was slightly narrower for green than red and blue. The left halves of field of vision, which were lost at first attack, still very imperfect; colour vision is completely absent, and he can only distinguish between light and darkness. In November, 1890, a portion of the inner lower quadrant of the right eye had recovered, and two (possibly overlooked) defects to the left of fixation point were discovered. The intactness of vision in the neighbourhood of the fixation point, in this case as in others, supports Foerster's hypothesis that the frequency of the deviation of the separation line towards the defective side is not due to a blending of the fibres of both optic tracts in each retina, but to a more favourable blood supply in the centres of occipital lobe which subserve the function of sharpest vision.

Colour-sense normal in the restricted field. The author remarks that this fact, compared with the absolute colour-blindness in Foerster's case, is perhaps due to the greater extent of the field in X.'s case, and confirms Foerster's contention that for the distinction of colour a large cortical surface is required. The latter must have been more impaired in Foerster's case. Colour-memory good; no phosphenes, but he experiences "round the eye a wave-like motion like water rippled by the wind." No aphasia; reads and writes well, both from memory and dictation.

Almost complete loss of the sense of locality. Everything since the second attack makes a strange impression on the patient; he does not recognise his own room, nor can he find his way about his house. If placed, however, in the corner of a room, and a door is pointed out to him, he goes directly to it (even with closed eyes), avoiding any obstacles. His power of orientation in the open is very defective. All streets appear much longer than formerly, but not so periods of time. Judging distances at first difficult, all objects appeared to be nearer than in reality, but soon corrected. His mental condition otherwise normal, except slight forgetfulness; quite conscious of his deficiencies. The different psychical functions related to the sight-sense also found fairly normal, except a considerable deficiency in the number of his visual memories. Older impressions better preserved. Fresh visual memories retained for some minutes only. Assisted himself by touch impressions. His geographical memory is practically gone.

In discussing the symptoms of this case, Dr. Groenow comes to the conclusion that the lost power of orientation and memory of locality is to be referred to this partial loss of visual memories. It is possible to acquire correct topographical impressions through the senses of touch and space, as well as by description, in cases where occipital lesions have destroyed the visual locality sense. A man, blind from peripheral lesion, could after a very few days find his way about a room guided solely by the sense of touch; this X. could not do. The presumption is that every representation of space acquired through touch excites simultaneously the corresponding visual representation, and that it is this secondarily excited visual picture which is mainly preserved.

The latest accounts of the patient show that he improved considerably while taking iodide of potassium, he gradually learnt to orientate himself better, but there was no alteration in the limits of the field of vision.

The conclusions that Dr. Groenow draws from this and the other cases referred to are :—

I. If in cases of bilateral hemianopsia of central origin one part of the field of vision remains intact, that this part contains as a rule (perhaps always) the fixation point.

II. The disturbances of sense of locality occuring in lesions of the occipital lobe rest upon the loss of numerous visual memories.

J. Turner.

Mahaim on the Fillet. (*Archiv für Psychiatrie*, vol. xxv. pp. 343-382.)

In a very exhaustive and fully illustrated account of a case of limited lesion on the right side of the cerebrum, the author draws conclusions with regard to the connections of the fillet which are at variance with those expressed by Flechsig, Hösel and others. According to him, the fibres of the fillet are not directly connected with the cerebral cortex, but terminate above, for the most part at least, in the optic thalamus.

The lesion, which was apparently an old softening, was limited to the supra-marginal and superior temporal convolutions, and to the white matter underlying the central convolutions; to the external division of the lenticular nucleus and the whole of the internal capsule, except a small portion posteriorly and the optic radiation from corpus geniculatum externum to the occipital lobe. In the optic thalamus the external nucleus was entirely absent, the internal nucleus showed a great diminution in its cells, and, while the internal medullary lamina was absent, the external medullary lamina and the lattice layer were present, but smaller than on the left.

Mahaim found that there was only partial atrophy of the median fillet of the same side, the inter-olivary stratum and the internal arcuate fibres of the opposite side, and not such an extensive disappearance of those fibres as should have been present if the view that the motor cortex is their nutritive centre is correct. From this Mahaim infers that the atrophy of the fillet was merely secondary to the disappearance of the cells in the optic thalamus, and, except to a limited extent, could not have resulted from the cortical lesion directly. The results obtained should be compared with those described by Hösel (in vol. xxiv. of the same *Archiv*), in which a somewhat more extensive lesion of the central convolutions was followed by a complete degeneration of

the median fillet, although the optic thalamus had remained intact.

It seems difficult to reconcile those opposing views if one can be satisfied that the method of staining adopted by Mahaim is sufficient to demonstrate completely that the internal capsule and cortex in his case, to which the fillet fibres are supposed to pass by Hösel, were completely destroyed. The writer of this abstract publishes a case of complete descending degeneration in the median fillet as a result of a lesion in the optic thalamus, the internal capsule and the lenticular nucleus, and he has under examination at present a case of extensive destruction of the cortex of the occipital lobe and post-central convolutions, in which the condition of the fillet appears closely analogous to that of Mahaim's case. These two cases would appear rather to support the view that the fillet terminates in one or other of the basal ganglia rather than in the cerebral cortex. It seems probable, from other considerations, that there is a trophic centre for the median fillet in the nuclei of the posterior columns as well as in its upper termination, and that, under certain conditions, yet to be determined, but which appear to have some relation to the youth of the individual, the former may long maintain its activity.[1]

ALEXANDER BRUCE, M.D.

Hammond on Progressive Muscular Atrophy.

DR. G. M. HAMMOND exhibited before the American Neurological Association, July, 1893, microscopical specimens, and gave the pathological report of two cases of progressive muscular atrophy, and referred to the fact that considerable confusion is occasioned by the misapplication of the term "peroneal type" to a disease totally dissimilar to the one under consideration.

1.—The first patient was a woman, forty-six years of age, without history of gout, rheumatism, or syphilis. In the fall of 1889, she stumbled over a chair, bruising the right shin slightly, and a few days afterwards she was unable to walk as well as formerly. Soon after, there was marked weakness of the flexors of the foot and the extensors of the toes.

By October, 1890, there was complete paralysis of the entire right side, with reaction of degeneration, loss of power and electrical contractility of the anterior tibial muscles of the left leg,

[1] (See criticism of Mahaim's views by Hösel: "In Sachen Rindenschleife" Neurol. Ctrblt. No. 17, 1893).

and atrophy and loss of power of the thenar and hypothenar muscles of the right hand.

Gradually, but rapidly, the other extremities became involved, and ultimately the abdominal and intercostal muscles were affected. She died suddenly from either cardiac or respiratory paralysis.

The sections of the cord showed sclerosis of the pyramidal tracts and atrophy of cells in the anterior horns, and degeneration of Gowers' column throughout its entire extent. The conclusion to be drawn from a clinico-pathological study of this case would be that the muscles of the hand were supplied by the mesial group, and the muscles of the forearm from the anterior group of cells in the anterior horn.

2.—In the second case, first seen in 1882, the disease began in the muscles of the left thumb. He had the opportunity to watch the case to its termination, in 1893. It ran the usual course, and was in every way typical. At the time of death all of the muscles from the level of the armpits upwards, except the facial muscles, were atrophied, as well as those of the upper extremities. She became maniacal, and finally died from exhaustion.

The cord and medulla were examined, and the changes were identical with those described in the preceding case.

He concluded that these cases demonstrate that progressive muscular atrophy is due to a degeneration of the cells in the anterior gray masses and the nerve tubes in the antero-lateral white columns. Also that it is superfluous to divide progressive muscular atrophy into different types because the disease happens to begin in different groups of muscles. (*New York Medical Journal*, January 6th, 1894).

Laycock on a Case of Brain Injury, with loss of Muscular Sense.

On 24th February last, while the patient was cutting down a tall gum tree, a branch about the thickness of a man's arm fell from the height of about 100 feet on his head. The patient was standing firmly in position for swinging his axe, and the falling branch meeting what was practically a rigid body, simply crushed in the skull, just behind the right parieto-frontal suture. The patient was admitted to the hospital next day. He was then unconscious, with great restlessness and delirium. The right eye was contused, the pupils were equal, and there were left hemiplegic symptoms. The vertex was extensively fractured,

and in the wound there were pieces of bone and brain substance. Mr. Cooke cut down over the wound and removed ten pieces of bone, the gap in the cranium being an irregular oval measuring four by three inches. He explored the wound with his finger, and found the base of the skull fractured just behind the orbits, there being a fissure about a quarter of an inch wide. The right frontal bone could be easily moved, and in fact the skull had been practically split in two in the line of the blow. A good deal of lacerated and contused brain substance was removed, the edges of the dura mater brought together as far as possible, and the wound closed.

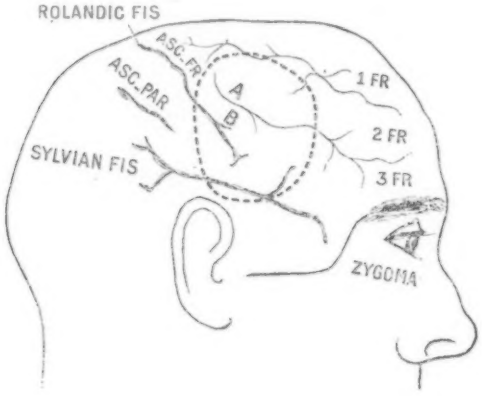
After the operation, the patient was very restless, extremely noisy in his delirium, and was continually throwing his right arm about. The nurse reported that he moved his arm in a circular direction, and that if anybody was speaking within earshot of him, he would catch up the last two or three words and repeat them several times. The hemiplegic symptoms were well marked. There was scarcely any rise of temperature.

On the sixth day after the accident, consciousness began to return, and he could recognise people about him. At this time there was noticed right facial paralysis; this, however, passed off in about four weeks. From this time he gradually became more rational, though very restless and excitable. On the tenth day a hernia cerebri began to threaten, but this soon passed off. The improvement continued, and in about a month the hemiplegic symptoms began to disappear.

During his illness he complained from time to time of a cramping sensation in the flexors of the right forearm and a slight pain in the part, but there was no rigidity or tenderness of any kind. He continued very emotional, but in every other respect the progress was continuous, and on the 4th of May, just ten weeks after the accident, he got up.

Laycock first saw him at this stage, and his condition was as follows:—He could walk with assistance, but in a very uncertain manner, his gait being distinctly hemiplegic. He had good average power of grasp in the left hand, and the muscular power in the left leg seemed unimpaired. Voluntary movements were performed with the left limbs in an extremely awkward manner. He had complete loss of sense of position in the leg, and incomplete in the arm. If his leg were moved when his eyes were shut, he could not tell where it had been moved to. Under similar circumstances he could tell the position of his arm, but with considerable uncertainty. He could recall in idea a series

of positions, the change being made with his eyes shut. He could move the muscles of his face, but could not make a specified movement. He said he could do it, but did not know how. The movements of the eyeball and eyelids were perfect, also those concerned in speech. The sense of weight was not tested, but the allied sense of resistance was almost absent. When his hand was pressed down, say on the bed, and he was told to move it towards his face, his eyes being shut, he was incapable of bringing the requisite amount of force in the proper direction, and made no difference in the amount of force he used on the pressure being



The dotted line shows the extent of the injury to the bone, the letters A and B are placed over the deepest part.

increased or decreased; also he was quite unable to disengage his hand from one pressing it down, and struggled in an aimless kind of manner. On the right side there was no symptom, except a crampy feeling in the right forearm. There was, however, no spasm or motor disturbance, and no loss of strength at that time. The right leg was quite unaffected. The knee-jerks and plantar reflexes were absent on both sides

As regards sensation: On the affected side he had sensation of a very rudimentary description, there being only tactile and painful sensibility. When he was touched he felt he was touched, but did not know what kind of thing had touched, or where

the point was in the limb. He stated that soon after he regained consciousness he felt a fly walking over the back of his hand, but he did not know that it was a fly, or where it was without looking for it. If his arm were touched, his eyes being shut, he marked the spot two or three inches below the point. If his arm were slightly pinched, say on the outside of the elbow, he felt the pinch in the fingers, or as he described it, between the ring and forefingers. If on the inner side of the elbow, the sensation was between the little and ring fingers. If the arm were strongly pinched the pain shot all over it, without being localised in any way. In the skin of the face there seemed to be a delay between the contact and the mental conception. The same result could be got from the arms, but not so marked. He could not distinguish heat from cold, but if the heat of the water were excessive, pain was caused which spread all over the arm. In the leg, the sensory disturbances seemed to run in the same lines, but the symptoms could not be so clearly brought out.

The depression on the head forms an irregularly-shaped oval, 9 cm. by 7 cm., its deepest points A and B in the diagram being 3.7 cm. and 3.5 cm. respectively, measured from the perpendicular plane to the external meatus. The posterior wall slopes abruptly to the deepest part, the anterior more gradually. The upper margin is 5.5 cm. from the middle line at a point 17 cm. from the nasion, and the lower margin is a similar distance from the zygomatic line. (For purposes of comparison, the dimensions of the patient's head are given in the middle line from the root of the nose to the occipital protuberance 36.2 cm. Laterally, between these two points, 28.75 cm. Between the external meatus 15.5 cm., from the external meatus to the middle line at the vertex 18.5 cm., cutting the middle line 17.5 cm. and the lateral line at 14.7 cm. from the nasion.)

The following parts of the cerebrum would appear to be involved:—The lower half of the ascending frontal convolution, and the greater part of the sigmoid flexure, the posterior third of the lower and middle frontal convolutions, and the base of the posterior end of the upper one; also the base of the corresponding part of the falciform lobe. The destruction being deep at the part the nerve fibres radiate from the anterior half of the internal capsule, these fibres must have been affected close to their emergence. Thus it will be seen that the motor centre for the arm would be the one principally involved. Those of the leg and trunk and of the face would be affected to a less degree.

As regards his other senses—hearing, taste, and smell are quite unaffected. Speech also, and the promptitude with which he answers questions, forms a marked contrast to the slow and apparently stupid manner in which he performs movements with his affected arm.

Ophthalmological examination.—Fundus rather pale; veins enlarged; arteries small; muscles of the eye, both external and internal, normal; vision, as far as it was tested, was quite normal.

His temper seemed quite unaffected, and his memory and intelligence were perfect. He is rather emotional, and inclined to laugh or cry. The rest of the body is sound and healthy, without the slightest indication of any disease.

As soon as the wound had completely healed up, and the patient was able to bear it, L. endeavoured to get some localising indications from electrical stimulus. The indifferent pole, a flat-electrode, 5 by 8 cm., was placed on the back of the neck, the other pole, with a small olive-shaped point, was placed on various parts of the depression. No result was obtained from the faradic current, the patient being able to bear only a very mild degree of strength. A galvanic current of 5 to 10 milliamperes yielded the following results:—With the anode in the inferior part of the posterior margin—the face centre—on each interruption of the current there was a sensation in the neck on the opposite side. With the kathode, the sensation was more localised, and went through both sides of the neck just above the level of the angle of the jaw. With the anode in the middle of the posterior margin—the arm centre—there was a sensation in the shoulder and upper part of the arm. With the kathode, the sensation extended down the arm in a slow crawling manner (to use the patient's expression), and when it got to the fingers there was a tingling as if the hand had been numb with cold, and had been brought to a fire. The patient was able to follow the sensation downward with his finger. On the current being increased to 12 or 13 milliamperes, both sides were affected; the right, however, not so much as the left, the sensation downwards being of much greater rapidity. On the anode being placed on the upper part of the posterior margin—the centre for the trunk and thigh—there was a sensation just at the angle of the neck and shoulder. With the kathode the sensation was in the same region, but somewhat deeper from the surface. In none of these observations was any muscular action obtained. The sensations were not painful, being only of a tingling character, and it should be

noted that the upward current gave far more definite results than the downward.

As regards the subsequent history, the patient has improved steadily in the condition of his left arm and foot, and performs movements with them much more expertly. The right arm, however, is showing signs of weakness. When L. last saw him, the crampy sensation in the flexors of the hand and wrist was still there, and it was accompanied by a burning pain at the same region. There was no tenderness or swelling, and no headache of any kind. The pain was best relieved by simple rubbing.

On reviewing the history, there are one or two points of interest. From the nature of the injury, a zone of brain substance just in front of the fissure of Rolando would bear the brunt of the blow, and its left half would, at least, be severely concussed. This left half would include Broca's convolution (the centre for speech), and the anterior end of the temporal lobe (one of the auditory centres). It will be remembered that it was noted that, for two or three days after the accident, the patient was very noisy, and if anyone were talking in his presence, he would catch up the last two or three words of the sentence and repeat them several times, he being unconscious at the time. This would signify that the auditory and speech centres were in a state of extreme excitability, and that the stimulation by the vibration of the sound of a word would set automatically into action the whole chain of centres, the result being the spoken words. The zone of brain substance would also include the centre for the right arm. During the first two or three days he threw about this arm a great deal, chiefly in a revolving manner, which showed its cortical centre to be in a highly irritable state. The symptoms of cramp and burning pain, and the weakness which has come on more recently, have certainly a causal connection with this, and it seems to me to be rather ominous, and shows that there is some mischief, probably of an inflammatory character, going on in the left frontal lobe.

The cerebral injury presents a problem of the greatest complexity. With the exception of a small part of the base of the falciform lobe, which is considered to be sensory, the bulk of the part destroyed is in the area which is generally regarded as motor. Nevertheless, the sensory and motor functions of, say, the arm, as it is the part most completely involved, seem to be affected in a relatively equal degree, and both of these functions seem to have been cut down to a very rudimentary level.

On the sensory, or to be more exact, the afferent side, there is simple tactile sensibility, but there is an absence or deficiency of the higher conceptions of sensation, which are evolved from the lower and simpler levels, such as the position of the limb, the qualities and position of foreign bodies touching the part, their weight, resistance, &c. Similarly on the motor or efferent side, there is the power of making simple movements, but there is an absence or deficiency of the power of making the more complicated ones, which must necessarily be on a higher developmental level.

As both these afferent and efferent functional disturbances are the result of one and the same injury, it follows that the part destroyed, that is, part of the Rolandic area, is sensori-motor. What, however, the comparative importance of each of these factors is, and where their functions end and begin, are the most difficult problems in neurology. These afferent impressions make up the complex which forms the so-called muscular sense, and as they are severely affected, especially in the arm in the case under discussion, the symptoms of loss of this sense are very well seen. In the group of impressions and sensations which form this sense, L. is inclined to regard the sense of position as of the greatest importance, and the awkwardness of movement in this case depends more upon this than upon anything else. The nicety of muscular movement, and the quantity of force used, entirely depend on a knowledge of the point from which the muscular force has to commence action, and of the successive changes of position or relations which the movement causes.

These estimations of position are always present in the normal body, as anyone can ascertain for himself, and it leads me to the belief that they are not of the nature of ordinary sensory impressions, such as pain or heat and cold, but that there must be some arrangement by which a circuit of nerve force is continually in equilibrium. In the cerebral part of this circuit there will be some sort of centre like a galvanometer in an electric circuit, which will keep the cortical centres informed, so to speak, of the state of the peripheral parts, not only when they are at rest, but when they are in action, action being indicated by change from one state of equilibrium to that of another. In our case, injury to the cortical loop of such a circuit could explain many of the symptoms.

As regards the sensory disturbance, here again the sense of position takes an important place. Common or cutaneous sen-

sation, with pain or some variety of pain, has its nervous tract in the posterior end of the hindmost limb of the internal capsule. This has escaped injury in our case evidently, for common sensation seems to be actually increased. The sense of tactile localisation is almost lost, and L. believes we may infer that this faculty is in a different nervous tract from common sensation. In the diagrams of the internal capsule, the anterior half of the anterior limb is left blank. From the experience gained by this case, L. thinks it may be fairly filled up by the tracts of the so-called muscular sense, or, as L. prefers to call it, the sense of local relation.—(Abridged from the *Australian Medical Journal*, July, 1893.)